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IN THIS ISSUE

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CONTENTS

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REGIONAL TICK PARALYSIS

SENSORY AND MOTOR CHANGES CAUSED BY A MALE TICK, GENUS HYALOMMA

L. D. Erasmus, M.B., B.Ch. (Rand)
Department of Internal Medicine, University of Pretoria, Pretoria

Tick paralysis has been known for more than a hundred years. It affects domestic animals and man, and has been widely studied in Australia and North America. It is rare in Europe, but has been reported in sheep from Yugoslavia and Macedonia [11, 13] and in a hedgehog in Great Britain.[25] In South Africa it was first found to occur in sheep in 1904, but only recently has a human case been reported.[28] Extensive reviews of tick paralysis have been published.[1, 19, 22]

In man, children are more commonly and more severely affected, and they may not infrequently die.[7] An ascending motor paralysis with diminished or absent tendon reflexes is usual. If bulbar paralysis ensues, the lower cranial nerves are involved first as the process moves upwards. Ocular palsies may occur with blurring of vision. Ataxia may be noted in the earlier stages, but sensory changes are usually absent. Parasthesiae in the areas of motor weakness have occasionally been mentioned.

Regional paralysis has been reported from Australia.[3, 5, 6, 7, 20] In 4 of these cases the tick had attached itself in the external auditory meatus or on the scalp, producing a partial or total facial paralysis. In one case, reported by Hamilton,[7] a tick bite in the axilla had led to paralysis of the dorsiflexors of the wrist, with paresis of the other muscles of the forearm and arm. Full recovery occurred 6 days after removal of the tick. In none of these cases were sensory changes found, nor was the tick identified in any.

The condition is probably not an infection, since it cannot be transmitted, nor has any infective agent been demonstrated. Paralytic signs are associated specifically with the final stages of engorgement of the tick, no matter how long after attachment this may occur.[16] Recovery is usually prompt after removal of the tick, and there is never any delayed subsequent onset of paralysis. It therefore seems likely that a toxin is responsible, especially as the symptoms in many ways resemble those caused by certain known toxic agents, in particular coniine and diphtheria toxin.[26, 27] The toxin is almost certainly contained in the salivary secretion of the tick but its nature and origin remain uncertain. A toxic agent has been demonstrated in the eggs of certain ticks [15] but other investigators have concluded that it is not the same as that causing tick paralysis.[4, 10]

Little is known about the pathology of tick paralysis. Ferguson [3] reported the findings in a male child, aged 16 months, who died of tick paralysis. He found intense engorgement of all parts of the brain and diffuse round cell infiltration without perivascular cuffing. Changes have been described in the spinal cord and medulla oblongata of experimentally paralysed dogs.[8, 16]

The tick causing paralysis in Australia is *Ixodes holocyclus* Neum, while in America *Dermacentor andersoni* Stiles is the chief culprit. American cases have also been ascribed to *Dermacentor variabilis* Say, *Amblyomma americanum* and *Amblyomma maculatum*.

Despite general similarities, the Australian and American cases differ in certain ways, and this has been attributed to the types of tick concerned. The American cases apparently recover within a few hours after the tick has been removed while in Australia the paralysis may progress to a maximum 48 hours after tick removal. Regional paralysis is also commoner in Australia.

In the single human case reported from South Africa,[28] an adult male Bantu exhibited stupor, general muscle weakness and a slow recovery over a period of 7 weeks. Drowsiness seems to have been marked. The cerebrospinal fluid pressure was raised to 300 mm. water and there were retinal haemorrhages and papilloedema. Attached to the lower abdomen was a tick, identified as *Rhipicephalus simus* Koch. There have been 2 previous case reports from Somaliland, North Africa, of paralysis suspected to be due to the same tick [24] but Stanbury and Huyck [19] found the evidence unconvincing. In the case report which follows, the features were quite unlike those of the South African case mentioned above.

CASE REPORT

The patient, a Msutu male farm labourer, aged 21, was admitted to Prof. H. W. Snyman's clinic at the Pretoria General Hospital on the fourth day of his illness.

On waking 3 days before, he had felt a burning pain in the right axilla which spread to the elbow. That afternoon the pain was worse and his right arm considerably weaker than the left. The following morning his grip in the right hand had become feeble and his arm so weak that he could scarcely lift it. The pain in the right axilla had

not altered, but the burning feeling in the arm had been replaced by a numbness (a total loss of sensation) along the medial side of the upper arm and the medial and volar surfaces of the forearm. He then discovered an engorged tick in the right axilla and snipped off its hinder portion to encourage spontaneous loosening of the head.

On admission he appeared a little drowsy, was obviously in considerable pain and supported his right arm with his left. In the right arm there was extreme weakness of about the same degree in the deltoid, triceps, biceps, the other flexors of the elbow, the pronators and supinators of the forearm, flexors and extensors of the wrist and fingers and the intrinsic muscles of the hand. No muscle below the shoulder was spared. The biceps, triceps, supinator and finger reflexes were all present and slightly, but distinctly, increased. Co-ordination of movement could not be tested because of the weakness.

All forms of sensation were entirely lost below the shoulder except for a small area about 3 cm. long by 2 cm. wide on the extensor surface of the upper arm and a strip on the extensor surface of the forearm, about 3–4 cm. wide, where sensation was preserved. The strip of normal sensation extended from just above the right elbow to the knuckles, widening out a little on the dorsum of the hand and appeared to correspond with the sensory distribution of the radial nerve.

The patient's temperature on admission was 99° F but fell to normal after 2 days. Nothing further of note was found clinically.

The *erythrocyte sedimentation rate* was 7 mm. per hour (Westergren). The *blood count* showed: Hb, 17.9 gm. per 100 c.c.; erythrocytes, 5,970,000 per c.mm.; leucocytes, 7,000 per c.mm., with a differential count of polymorphs 49%, lymphocytes 40%, monocytes 7% and eosinophils 4%. The *cerebrospinal fluid* was clear, the pressure 100 mm. water, and the Queckenstedt test showed a rapid rise and fall of the fluid in the manometer. The protein was 43 mg., chloride 700 mg. and sugar 75 mg. per 100 c.c. There was 1 lymphocyte per c.mm. and the Wassermann reaction on both the fluid and the blood was negative.

The shrivelled remains of the tick were removed from their attachment in the right axilla. A tiny, punctured bite wound, with no obvious surrounding inflammatory reaction or regional lymphadenopathy, was revealed. The skin was exquisitely tender within an inch round the bite. Dr. G. Theiler identified the tick as a *male* of the species *Hyalomma transiens*.

The site of the bite with an area of surrounding skin was excised and serial sections, stained with Giemsa's stain, were prepared. These showed an epidermal opening beside a hair follicle which continued down into the corium as a serrated shaft. This shaft ended opposite the hair root, where it was surrounded by a broad area of liquefaction necrosis. In and around this area the blood vessels were dilated and necrotic. There was considerable infiltration with polymorphs, but eosinophils were absent. In the neighbourhood of the bite shaft the skin vessels showed endothelial swelling with perivascular oedema and lymphocytic infiltration. A small cutaneous nerve accompanying one such vessel was pale and swollen where the vascular reaction was greatest, but resumed normal appearances on either side of this zone. Cocci were abundant in the hair follicle openings, but in the depths of the bite only a few coccal clusters were seen.

Progress. Two days after admission all muscle groups had recovered a fair amount of their power. The triceps and extensors of the wrist and fingers remained the weakest. The pain in the axilla was less intense. The area of total sensory loss had shrunk considerably and was absolute only in an area about 5 cm. broad, extending from just below the axilla to the wrist along the medial border of the arm and forearm. The area of tactile sensory loss was about 2 cm. broader than the area of loss of pain and temperature sense. There was still relative dulling of sensation in the area of previous sensory loss. The area of anaesthesia at this stage appeared to correspond with the distribution of the medial brachial and antebrachial cutaneous nerves.

Five days after admission there was normal power in all the muscles except the triceps, which still showed a mild paresis, but it, too, recovered completely within the next 2 days. The area of anaesthesia diminished from above downwards and 3 weeks after admission there remained only a small area of sensory loss below the right elbow on the inner surface of the forearm. This was still present 3 months after discharge.

In this case it was questioned whether the presence of the tick might not have been co-incidental in a patient suffering from pressure paralysis, a not uncommon condition following alcoholic stupor. However, the local pain, the development of weakness while the patient was ambulant, and the progressive motor and sensory changes over a period of days is quite unlike pressure paralysis.

FURTHER EVIDENCE OF REGIONAL TICK PARALYSIS IN SOUTH AFRICA

i. The author interviewed a European labourer, aged 52, who, 30 years ago on a farm 80 miles west of Pretoria, had experienced anaesthesia of a large area of his arm following attachment in the axilla of a *bontpoot* tick, almost certainly *Hyalomma transiens*. Severe motor weakness of the whole arm occurred. Recovery was complete within a few months after detachment of the tick.

ii. Four years ago, a 4-year-old European child in the Willowmore district of the Eastern Province was found by Dr. S. P. Perold[14] to have a total sensory loss along the lateral border of the right arm and hand, together with a marked wrist drop and weakness of other arm and forearm muscles. Recovery was complete 3 months after removal from the axilla of a partially engorged tick, species *Ixodes rubicundus* Neum. Paralysis in sheep due to this tick is rife during the late autumn in the Willowmore district.

iii. Undoubtedly motor weakness of the hind leg of his setter dog has been noted by a physician, Dr. G. H. Roux,[15] formerly a zoologist and now on the staff of this hospital. He identified a tick found between the dog's toes as a *bontpoot*, again almost certainly *Hyalomma transiens*.

DISCUSSION

The special features of the case described are that the paralysis was due to the bite of a male tick of the genus Hyalomma, with the development of intense and persistent sensory changes.

Male ticks have not so far been implicated. Ross,[17] referring to *Ixodes Holocyclus*, states: 'The male seldom attaches itself to a host and is of no pathogenic importance.' Most observers maintain that the bite of a gravid female tick is necessary to cause paralysis in man and animals. There is at least one case report, however, in which paralysis was caused by a non-gravid female.[12] In animal experiments nymphal and larval forms have also been shown to cause paralysis.[13]

It seems that an essential factor determining whether

a tick will produce paralysis is the rate and stage of its engorgement, rather than the sex. Paralysis appears to occur only in the terminal stages of engorgement of a rapidly feeding tick, which then injects a large dose of toxin into its host. The gravid female, above all, engorges rapidly and maximally. The male, on the other hand, is generally a poor feeder. In the present case, however, the male tick involved appears to have become quite as swollen as the average female.

Hyalommas have been known to cause paralysis in sheep in Yugoslavia,[11] and necrotic ulceration of the skin in dogs resulting from the bites of these ticks has recently been described.[23] Although they are very widely distributed in South Africa, Hyalommas have never before been implicated as a cause of paralysis or any other tick-borne illness in man. They have not been known to cause or transmit any disease in animals either, in this country, except for skin ulceration in dogs. This is not surprising since the hosts of the larval and nymphal stages are chiefly birds which feed and nest on the ground. These immature stages are seldom found on mammals. *Hyalomma aegyptium* (? transiens) has, however, been suspected of causing both local and generalized types of paralysis in lambs in the Rift Valley of Kenya.[9] Dr. G. Theiler comments as follows on the relationship between the Hyalommas and other ticks known to have caused paralysis in man or animals: 'Of all the species implicated, those belonging to the genus Ixodes are the most primitive. The Rhipicephalinae as a sub-family are more advanced than the Ixodinae. Of the Rhipicephalinae the two most highly developed genera, Boophilus and Margaropus, have not yet been involved in tick paralysis, nor has one of the two most primitive genera, Aponomma. The other, Amblyomma, has recently been implicated in America.[12, 21] The intermediate genera ranging from the primitive Haemaphysalis to the advanced Rhipicephalus have all been implicated. The genus Hyalomma has its greatest affinities, according to present-day workers, with the genus Rhipicephalus.'

The sensory changes in the case described were of an order not previously recorded in a case of tick paralysis. Bruce,[2] however, in 1920, mentioned 2 cases in which 'certain parts of the legs and arm had lost all sense of feeling', where the tick responsible had, presumably, been *Dermacentor andersoni*. The persistence of sensory changes long after the motor weakness had recovered was noteworthy in the present case. The site of attachment of the tick in the axilla was very close to the course of the two sensory nerves most affected, the medial brachial and antebrachial cutaneous nerves, although they are normally deep to the deep fascia at this point. It seems probable that they received the maximum concentration of toxin.

Of the muscles involved those supplied by the radial nerve were the last to recover. It is not known why the sensory portion of the radial nerve escaped. Perhaps it ran an abnormal course in this case.

SUMMARY

1. Tick paralysis is known commonly to occur in domestic animals in Australia, on the American and African continents and in parts of Europe and Asia. Human cases have been reported from the North American continent, Australia and North and South Africa.

2. The disease usually takes the form of an ascending motor paralysis which may be fatal. Children are the chief victims. The condition is probably due to a neurotoxin injected with the saliva of an engorging tick. Cases of regional paralysis have been reported previously only from Australia. The American cases recover sooner after removal of the tick.

3. A case is reported from South Africa, in which a tick bite in the axilla caused extensive, regional, sensory and motor changes in the corresponding arm. Recovery occurred after removal of the tick. The clinical features were quite unlike those of the case of tick paralysis reported from this country.

4. A male *Hyalomma transiens* was the tick involved. It has not before been known to cause paralysis in man, nor has a male tick previously been implicated.

5. Evidence which suggests that *Ixodes rubicundus* Neum may cause local paralysis in man and further evidence implicating *Hyalomma transiens*, is presented.

6. It is suggested that the stage and rate of engorgement are more important for the production of neurotoxin than the sex of the tick.

7. Histological studies of the skin in the region of the bite revealed local inflammatory changes but no gross alteration in related nerve fibres.

I wish gratefully to acknowledge the assistance of Dr. G. Theiler and Prof. C. Jackson of the Veterinary Research Institute at Onderstepoort, as well as that of Dr. G. H. Findlay with the skin histology.

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VAN DIE REDAKSIE

DIE BIOLOGIE VAN DIE KLEUR VAN DIE MENSLIKE VEL

Die kleur van die normale mensvel hang van menigvuldige faktore af, waarvan sommige oorerflik is en andere van die omgewing afhang, waarvan almal nie presies bekend is nie. Basies bestaan die kleursel uit 'n mengsel van melanien en karoteen wat deur die kleur van die vel self en die aanwesigheid van bloed in die vlak haarvate verander is. Boonop beheer 'n groep metaboliese faktore, op 'n swak begrepe wyse, die omvang en kwaliteit van velkleursels, b.v. die bynierskors en moontlik vitamine C. Origens word die graad van kleursel van die vel wel deeglik deur blootstelling aan sonlig verander, veral in die geval van die nasies met ligter velle. Dit hang natuurlik gedeeltelik af van die vermoë om bruin te brand, 'n proses waardeur ultraviolet strale die kleurselveranderings in die vel stimuleer, mits die betrokke persoon geneties in staat is om bruin te brand. Albinos en sekere tipes van blonde mense is b.v. nie in staat om onder enige omstandighede bruin te brand nie, terwyl donker mense oor die algemeen meer geredelik as ligte mense bruin brand.

Hoewel dit redelik seker is dat velkleur geneties bepaal word, is dit gladnie seker hoeveel pare gene die kleurvoorkoms beheer nie. Davenport[1] het ongeveer 40 jaar gelede toe hy 'n vindingryke teorie voorgelê het, velkleur toegeskrywe aan 2 hoofpare gene wat hulle uitwerking baie op dieselfde manier teweeggebring het as die beroemde ertjies van Mendel se klassieke eksperiment wat geel en rond of groen en gerimpeld was. Op die basis van die 2-paar gene teorie, gelokaliseer op 2 verskillende pare chromosome, is 9 verskillende genetipes moontlik en derhalwe, teoreties, 9 fenotipes, d.w.s. uiterlike sigbare karakters. Davenport het aan die verskillende genes die mag om verskillende grade van swartheid voort te bring, toegesê. As die swart homosigoot as *AABB* beskryf word en die wit homosigoot as *aabb*, het hy die volgende grade van swart aan die verskillende genes toegeskrywe: *A*, 19%; *B*, 16%; *a*, 2% en *b*, 1%. Die volbloed Neger (*AABB*) sal op dié basis 'n 70% graad van swart bereik, terwyl die witvel-Europeaan 'n 6% graad van swart bereik. Die baster (*AaBb*) sal 38% swart wees. Hierdie interessante onderstelling is egter nie bevestig nie. Bowendien as al die moontlikhede uitgewerk word, sal dit opgemerk word dat hoewel daar teoreties 9 herkenbare

1. Davenport, C. B. (1913): *Heredity of Skin Color in Negro-White Crosses*. Carnegie Institute of Washington, Publication No. 188, bl. 1—106.

EDITORIAL

THE BIOLOGY OF HUMAN SKIN COLOUR

The colour of normal human skin depends upon a multiplicity of factors, some of them hereditary and some of them environmental, not all of them precisely known. Basically, the coloured appearance is made up of a mixture of melanin and carotene modified by the colour of the skin itself and the presence of blood in the superficial capillaries. In addition, a group of metabolic factors controls, in an ill-understood way, the extent and the quality of cutaneous pigmentation, e.g. the adrenal cortex and possibly vitamin C. Moreover, especially in the fairer-skinned peoples, exposure to sunlight profoundly modifies the degree of pigmentation of the skin. This, of course, depends partly on the ability to tan, a process whereby ultra-violet rays stimulate pigmentary changes in the skin, provided the person concerned is genetically capable of tanning. Albinos and certain types of blondes, e.g. are unable to tan in any circumstances, whereas brunettes generally tan more readily than blondes.

Although it is fairly certain that skin colour is determined genetically, it is by no means certain how many pairs of genes control the pigmentary appearance. Davenport,[1] some 40 years ago, propounding an ingenious theory, attributed skin colour to 2 major pairs of genes, which produced their effects in much the same way as the famous peas of Mendel's classical experiment turned out to be yellow and round or green and wrinkled. On the basis of the 2-pair gene theory, located on 2 different pairs of chromosomes, 9 different genotypes are possible and therefore, theoretically, 9 phenotypes, i.e. externally observable characters.

Davenport assigned to the different genes the power of producing different proportions of blackness. If the black homozygote is described as *AABB* and the white homozygote as *aabb*, he assigned the following proportions of black to the different genes: *A*, 19%; *B*, 16%; *a*, 2% and *b*, 1%. The full-blooded Negro (*AABB*) would, therefore, add up to 70% black in colour, whereas the pale-skinned European would add up to 6% of blackness. The mulatto (*AaBb*) would be 38% black. This interesting hypothesis has, however, not been confirmed. Moreover, when all the possibilities are worked out, it will be seen that although there are theoretically 9 recognizable phenotypes, in

1. Davenport, C. B. (1913): *Heredity of Skin Color in Negro-White Crosses*. Carnegie Institute of Washington, Publication No. 188, pp. 1–106.

fenotipes is, sal daar in die praktyk slegs 5 herkenbare kleurskakerings wees.

Dit is natuurlik onwaarskynlik dat daar net 5 tinte is wat die een uiterste van die ander skei, en dis derhalwe waarskynlik dat meer as 2 paar genes betrokke is by die bepaling van velkleur. As daar b.v. 3 paar genes op 3 verskillende pare chromosome betrokke was, sal daar 27 genotipes wees, en derhalwe, teoreties, 27 fenotipes, d.w.s. teoreties kan daar 27 verskillende kleurskakerings wees, en die kanse dat 'n ‚suiwer' witte of ‚suiwer' swarte uit baster ouers gebore word, sal 1 : 64 wees (teenoor 1 : 16 op die basis van 2 paar genes).

Davenport se werk was op Jamaika uitgevoer en dit volg nie dat die erflikheidsleer van velkleuring onder Jamaikane dieselfde is as onder ander mense-groepe nie. Volgens Davenport se teorie moet dit b.v. moontlik wees vir baster ouers om kinders wat donkerder en ligter as hulle self is, voort te bring. Dit behoort feitlik moontlik te wees, hoewel uitsonderlik, dat 'n algehele ‚swart' of algehele ‚wit' kind uit sulke gekleurde ouers gebore kan word. Die inwoners van Jamaika was egter van mening dat so 'n terugaarding waarskynlik 'n mite is.[2] Hierbenewens word die posisie gekompliseerd deur die moontlikheid van onwettige afstammelinge. Wat egter op die basis van genetiese bepaling van velkleur volkome duidelik is, is dat wanneer (van Gekleurde ouers) 'n kind met 'n ligte vel gebore word met 'n velkleur onuitkenbaar van dié van genetiese ‚suiwer' Kaukasiërs, dan is die velkleur in werklikheid net so suiwer Kaukasiërs soos dié van die oorspronklike Kaukasiese voorouer.

Velkleur en vorm van hare staan nie in verband met mekaar nie en hierdie eienskappe word derhalwe onafhanklik oorgeërf. Nou en dan sal kinders wat geredelik kan deurgaan as 'n volbloed indiwidu van die een of ander ras wat saam gevoeg is om dit voort te bring egter uit baster ouers gebore word. In sulke gevalle is die skyn werklikheid, en is die karaktertrekke wat Kaukasies voorkom in werklikheid geneties suiwer Kaukasies.

Baie min is bekend omtrent die menslike chromosoomkaart. Ons kan nie met sekerheid sê watter ander fisiese karakters met die chromosome verbind is wat die genes dra wat velkleur bepaal nie. Dit mag wees dat as gevolg van 'n basterkruising daar kinders gebore word met ‚Kaukasiese-velle' en ‚Neger-niere'. Dit kan nie voorspel word nie. 'n Begrip van die onderliggende beginsels en arbitrêre veronderstellings wat die karaktertrek van 'n ras bepaal, is nodig, maar die wyse waarop velkleur oorgeërf word, behoort duidelik te toon hoe gevaarlik dit is om jou te verlaat op die leek se konsepsie van hoe hierdie dinge bepaal word. Daar is te veel subjektiewe vooroordeel in verband met hierdie sake, en voldoende en doeltreffende navorsing in verband met hierdie belangrike probleem sal nie moontlik wees voor vooroordeel nie uitgeskakel is nie, sodat 'n objektiewe herberaming, verkieslik deur die gebruik van fotospektrometriese metodes, gemaak kan word nie. Dit sal 'n gesonde basis vir moderne fisiese antropologie voorsien en die belaglike bewerings van die leukoofiliese melanofobe ontbloot.

2. Boyd, W. C. (1950): *Genetics and the Races of Man.* Oxford: Blackwell Scientific Publications.

practice there would only be 5 recognizable shades of colour.

It is, of course, unlikely that there are only 5 hues separating the one extreme from the other, and it is therefore probable that more than 2 pairs of genes are involved in the genetical determination of skin colour. If, e.g. 3 pairs of genes on 3 different pairs of chromosomes were involved, there would be 27 genotypes and, therefore, theoretically, 27 phenotypes, i.e. there could theoretically be 27 different shades of colour, and the chances of a 'pure' white or a 'pure' black being born to mulatto parents would be 1:64 (as opposed to 1:16 on the basis of 2 pairs of genes).

Davenport's work was carried out in Jamaica and it does not follow that the genetics of skin pigmentation amongst Jamaicans is the same amongst other human groups. According to Davenport's theory, e.g. it should be possible for mulatto parents to produce children both darker and lighter than themselves. In fact, it should be possible, though rare, for a completely 'black' or a completely 'white' child to be born from such Coloured parents. The inhabitants of Jamaica, however, were of the opinion that such a 'throw-back' was probably a myth.[2] The position, moreover, is complicated by the possibility of illegitimate offspring. What is, however, perfectly clear on the basis of the genetical determination of skin colour is that when (from Coloured parents) a light-skinned child is born with a skin colour indistinguishable from that of genetically 'pure' Caucasians, the skin colour is as Caucasian in its 'purity' as that of the original Caucasian ancestor.

Skin colour and hair form are not correlated and these traits are therefore inherited independently. Occasionally, however, children will be born of hybrid parents who will be able to pass readily as a pure-blooded individual of either race which united to produce it. In such cases, things are what they seem to be and the characters which look Caucasian are indeed genetically pure Caucasian.

Very little is known about the human chromosome map. We cannot be certain what other physical characters are linked with the chromosomes carrying the genes determining skin colour. It may be that, as a result of a mulatto cross, children are born with 'Caucasian' skins but 'Negro' kidneys. There is no way of telling. An appreciation is necessary of the underlying principles and arbitrary assumptions determining the features of race; but the way in which skin colour is inherited should make clear the dangers of relying on the layman's concept of how these things are determined. There is too much subjective bias in these matters, and adequate and effective research into this important problem will not be possible until prejudice has been eliminated so that an objective reassessment can be made, preferably by the use of photospectrometric methods. This will provide a sound basis for modern physical anthropology and expose the ridiculous allegations of the leucophilic melanophobes.

2. Boyd, W. C. (1950): *Genetics and the Races of Man.* Oxford: Blackwell Scientific Publications.

TWO CASES OF STEVENS-JOHNSON SYNDROME

B. N. FRASER, M.B., B.CH. (RAND), M.R.C.P. (ED.)
Department of Medicine, King Edward VIII Hospital, Durban

Stevens-Johnson syndrome has been reviewed recently by Ashby and Lazar.[1] They analysed 77 cases taken from medical writings and 4 cases of their own. Their findings are summarized briefly as follows:

Age. Most cases (85%) occurred under the age of 30 years.
Sex. Males predominated (81%).
Season. Incidence was mainly in winter.
Prodromal Symptoms. In just under half the cases (47%) the skin lesions were preceded by symptoms such as fever, sore throat, cough or coryza of 1–13 days' duration.
Pyrexia. 96% of patients had pyrexia, usually reaching 103° F or higher.
Mouth Lesions. All patients had lesions of the mouth or lips. These lesions consisted of a grey or white membrane, which stripped to leave a raw ulcerated area, vesicles, erythematous macules or redness. In 42% of cases, the lips were crusted with blood, an appearance considered pathognomonic of Stevens-Johnson syndrome. Foetor oris and dysphagia were common.
Eye Lesions. In 81% of cases, there was involvement of the conjunctiva. Purulent conjunctivitis was commoner than catarrhal, but there occurred also membranous conjunctivitis, and bullae on the conjunctiva. In a few cases the cornea was involved.
Genitalia. In just over half the cases (52%), lesions of the genitalia occurred. In the male these took the form of ulceration on the glans penis near the meatus, ulceration on the scrotum or urethral discharge.
Skin Involvement. Skin lesions occurred in 83% of cases and were erythematous, vesicular or bullous in nature. Erythemata occurred in many forms. Vesicles or bullae were usually superimposed on erythematous patches and their contents were usually serous, but occasionally became haemorrhagic or purulent. The distribution of the rash was very variable, but the scalp was never involved. Recurrent crops were the rule.
Lung Lesions. 31% of patients had pneumonia, the radiological appearance of which was that of one or more patchy shadows.
White Cell Count. Leucocytosis was common and sometimes marked.
The course was 8–69 days, the majority of cases being discharged during the fourth and fifth weeks of hospitalization.
Recurrence. One or more recurrences occurred in 22% of cases.
Mortality was 11%.

The aetiology of the syndrome is not yet known. Factors suggested as causing the lesions include virus infection and various allergins, of which perhaps the most important are the sulphonamide drugs.

Treatment with Aureomycin has been reported by Church (1 case),[2] Lynas (1 case),[3] Loewenthal, Marais and Ruskin (2 cases),[4] Persky (1 case),[5] and Nellen and Lang (1 case).[6] In all 6 cases rapid improvement with recovery took place.

Below are reported 2 further cases in which Aureomycin was used with a favourable outcome.

Case 1. V. N., an Indian male aged 20 years, was admitted on 28 September 1951 with a history that 2 weeks before a sore had developed on the upper lip and then on the lower lip. This was followed by pain in the mouth and tongue and difficulty in speaking and swallowing. Two days before admission the inner aspect of the left eye had become painful as well. He stated that he had attended several doctors without success. There was nothing else relevant in the history.

On examination he was obviously ill. The temperature was 100.4° F and the pulse rate 88 per minute. Both lips were swollen and crusted with dry blood. He had difficulty in protruding his tongue, which was swollen and very tender. The oral mucous membrane was congested with patches of deeper erythema. Foetor oris was present. The inner aspect of both eyes showed inflammation of the conjunctiva with a bead of pus in the left inner canthus. There were no skin lesions or urethral discharge. The lung fields were clear on physical examination. X-ray of the chest was normal and the blood count revealed 4,200 white cells per c.mm. with 48% neutrophils, 42% lymphocytes, 5% monocytes and 5% eosinophils. Treatment with Aureomycin 500 mg. 6-hourly was begun on the morning after admission. Steady improvement occurred and by the fourth day the temperature was normal, the conjunctivitis had resolved and he was talking and swallowing easily. He was discharged on the eighth hospital day, by which time the lesions had completely disappeared.

Case 2. S. N., a Zulu male aged 26 years, was admitted on 8 May 1952. He said that 3 days before his mouth and throat had become painful. The day afterwards his eyes had become painful and a lesion had developed on his penis, which had caused bleeding. He stated that he had had 3 similar attacks before the present one, the first in 1948 (which had lasted 2 months) and the other 2 in 1949 and 1950 (both of which lasted 3 months). Like the present attack, these other attacks had occurred at the beginning of winter.

The patient was drowsy and obviously very ill, with a temperature of 103° F and a pulse rate of 96 per minute. His general appearance was not unlike that of a case of typhoid fever and, in fact, he had been admitted with that diagnosis. Foetor oris was marked. There were white membranous patches over the whole mouth and in some places these had stripped to leave raw areas. The lips also showed raw areas, but no blood crusts, possibly because the patient spat continually. Angular conjunctivitis with a slight purulent discharge was present. There was a fairly heavy crop of vesicles, up to 12 mm. in diameter, on the skin. The content of the vesicles was serous and half of them were in the centre of erythematous patches. The most heavily involved areas were the extensor surfaces of the upper limbs, but the flexor surfaces of the upper limbs, the trunk and legs were also involved. The palms, soles, face and scalp were spared. On the glans penis, surrounding the urinary meatus, was a large shallow ulcer. A purulent urethral discharge was also present. The lung fields were clear on clinical examination. The white cell count was 8,100 per c.mm. with 68% neutrophils, 24% lymphocytes and 8% monocytes. Direct examination of a urethral smear showed many pus cells and very few gram-positive bacilli. Culture was sterile. A throat swab was negative for *S. vincenti, B. fusiformis* and *C. diphtheriae*. Direct examination showed mixed bacteria. The Wassermann reaction was negative. Stool examination showed ova of Taenia and *Trichuris Trichuria*, but was otherwise normal. X-ray of the chest showed infiltration of the right upper zone with early changes in the left second interspace, the appearances being characteristic of early pulmonary tuberculosis. Sputum examination revealed the presence of acid fast bacilli, morphologically resembling *M. tuberculosis*.

He was treated with antiseptic mouth washes and 12-hourly intramuscular injections of Penicillin 500,000 units from the day of admission until 12 May, but without any noticeable improvement in his general condition or change in the lesions. The pain and discomfort continued unabated and the temperature ranged between 100° F and 103.4° F. On the afternoon of 12 May Penicillin injections were discontinued and Aureomycin 500 mg. 6-hourly was started. By 14 May he was noticably better, the vesicles were less tense and the temperature had begun to subside towards normal. On 15 May the temperature was normal, and the conjunctivitis and urethral discharge had disappeared. By 18 May the rash had dried up completely. When he was informed on 23 May that arrangements had been made to transfer him to a sanatorium at a later date, he discharged himself from hospital against medical advice. At that time, the rash had begun to fade.

DISCUSSION

It would be unwise, in assessing the effect of treatment in a disease so variable in severity and course, to decide that any one case had derived benefit from any one line of treatment. However, by now several cases of Stevens-Johnson syndrome have been treated with Aureomycin, and in all a steady improvement with disappearance of the lesions has ensued.

In the second case described here, it is significant that whereas all previous attacks had lasted 2-3 months, the last attack was limited to less than 3 weeks. It is a pity that more details of these earlier attacks are not available. The patient stated that he was treated in Port Shepstone Hospital on each of the 3 previous occasions, but despite extensive search of the hospital records the hospital authorities were unable to find any evidence that he had attended there. It is possible that he was admitted there under a different name. Alternatively he may have been treated at a mission hospital or clinic in the area.

The good results obtained with Aureomycin therapy may well be coincidental, but are nevertheless very suggestive, and it would appear that Aureomycin should be the first line of treatment in this condition. The successful outcome of Aureomycin treatment, where it has been tried, is further evidence that some of these cases at any rate are due to a virus infection.

SUMMARY

Two cases of Stevens-Johnson syndrome in non-Europeans are described. Rapid improvement with disappearance of the lesions followed Aureomycin therapy, confirming the good results obtained by other authors.

My thanks are due to Dr. J. L. Parker, Medical Superintendent, King Edward VIII Hospital, for permission to publish these cases and to the Radiological Department, King Edward VIII Hospital, and to the Provincial Laboratory for X-rays and laboratory investigations.

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CONGENITAL ICHTHYOSIFORM ERYTHRODERMIA

DESCRIPTION OF A CASE AND DISCUSSION OF THE RELATIONSHIPS OF SOME BULLOUS DERMATOSES

JAMES MARSHALL, M.D.
University of Pretoria, Pretoria
and
J. M. MARTIN, B.Sc., M.B., Ch.B.
South African Institute for Medical Research, Johannesburg

Congenital ichthyosiform erythrodermia (C.I.E.) is a relatively rare familial dermatosis. The case to be described is a typical example of the condition in its generalized, bullous form. The histological picture shows features which have not previously been studied in detail, and resembles that described in familial benign chronic pemphigus (Gougerot-Hailey [1, 2]) (F.B.C.P.). It has been suggested in the past that the bullous form of C.I.E. has histological features in common with epidermolysis bullosa,[3] but it has never been suggested that it bore any resemblance to F.B.C.P. There has been some controversy over the status of F.B.C.P. in the United States where most of the cases so far described (under a variety of names) have been discovered. Some observers believe it to be a bullous variant of Darier's disease,[4, 5] others a variant of epidermolysis bullosa.[6]

CASE HISTORY

The patient, a European boy aged 2 years, was born in South Africa. Both his parents originate from families resident for several generations in South Africa. No other member of the family in any generation is known to have suffered from any skin disease or to have any inherited defects.

The child had 2 small blisters on his face at birth, and others appeared on his buttocks when he was a few days old. Other crops of blisters appeared later. The skin was, at first, otherwise normal in appearance, but there gradually appeared a generalized scaliness which has varied in severity but has never entirely disappeared. The frequency of appearance of blisters has decreased with age. A great number of remedies has been used without the slightest effect.

When first seen, the whole body surface was abnormal in appearance. The main feature was a marked hyperkeratosis varying in degree from a gross verrucous thickening about the wrists and ankles to a flat scaly desquamation of the face (Fig. 1). A milder degree of verrucous hyperkeratosis was visible at the large flexures and on the abdomen. The body and limbs elsewhere were covered with large flat scales or plaques of grumous material. The palms and soles were grossly thickened. The hair on the scalp was matted with large thick greasy scales. There was a marked continual exfoliation.

The scales varied in colour from dark grey in the verrucous areas to fawn or yellow. The underlying skin was normal in colour except in the axillae and around the neck where there was a slight but definite erythrodermia.

No tense bullae were seen during the period of observation, but around the axillae and groins there was the

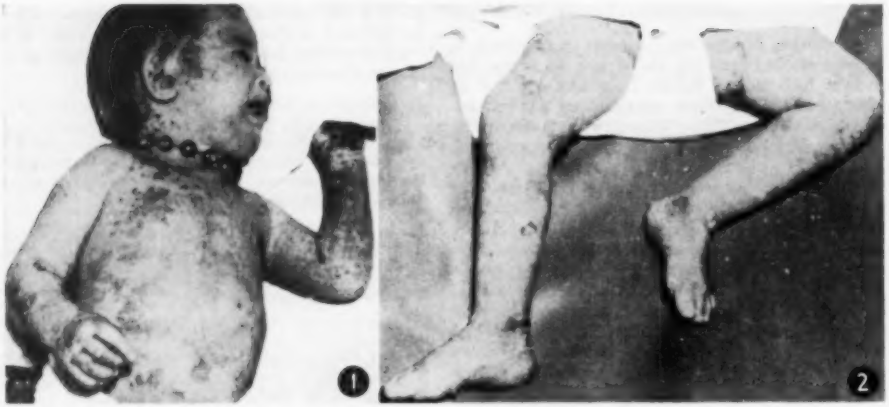

Fig. 1. Congenital ichthyosiform erythrodermia, showing generalized hyperkeratosis and scaling.
Fig. 2. Congenital ichthyosiform erythrodermia, showing hyperkeratosis and flaccid bullous lesions in the groins.

appearance of ruptured flaccid bullae, reminiscent of pemphigus foliaceus (Fig. 2). On the neck and back was a large area of eroded skin.

Nikolsky's sign was positive: friction on any part of the trunk or limbs rolled off the superficial layers of the epidermis leaving a moist, smooth, pink surface. Such a denuded area became, in a few days, even more heavily keratinized than the surrounding skin.

There was sometimes a mild intertriginous pyodermia about the ears and axillae which responded rapidly to treatment. Itching did not appear to be severe. The general health seemed little affected and the child was mentally alert. The hair and nails were normal in appearance and rate of growth, and there was no hyperhidrosis. The milk teeth, palate and ears were normal and the mucous membranes were not affected. The urine was always normal in colour and contained no excess of porphyrins. The blood picture was normal except for an absolute lymphocytosis, and serum tests for syphilis were negative.

The child was treated in hospital for six weeks. Vitamin A in large doses during the whole period and a 10-day course of ACTH had not the slightest effect on the condition. He was seen again about a month after discharge from hospital, when ruptured bullae were seen at the elbow flexures; there was also an impetigo of the face, scalp and neck which responded rapidly to treatment with antibiotics when he was readmitted to hospital.

Histology. The first specimen of skin examined was excised from the edge of a ruptured bullous lesion at the base of the neck. The epidermis shows hyperkeratosis, patchy parakeratosis and prolongation of the rete pegs. The stratum granulosum is not well marked and in some areas is completely absent. Irregular intra-epidermal bullae are present. The base of these bullae consists of the basal layer, which is completely intact, and 2, in places 3, layers of Malpighian cells. The roof is formed by a few layers of flattened, sometimes parakeratotic cells, to which groups of cells of the Malpighian layer are attached.

For the greater part the cells of the Malpighian layer have lost their intercellular bridges and have become ovoid and separated. These acantholytic cells lie suspended in the cavity either as single cells or in small clumps. Some of them stain normally, others are more acidophilic and appear hyalinized. This change is most marked in the single cells. The cells are slightly larger than the attached cells of the stratum mucosum, and have oval, well-stained nuclei with distinct nucleoli. An occasional nucleus is hyperchromatic. The cytoplasm in some of the detached cells shows a peculiar darkening around the periphery. The nuclei in these cells stain in a patchy fashion. Small masses of dark brownish material can be seen lying free in the cavity, presumably representing cytoplasmic rests from these cells which have broken up. In some places there is a structureless eosinophilic background in which red blood cells and small clumps of granular precipitate are present in addition to the epidermal cells.

There is no definite evidence of keratinization of the individual cells. *Corps ronds* have not been observed. The corium shows a mild, mainly perivascular infiltrate. The elastic tissue appears normal (Figs. 3, 4).

A second specimen of skin was taken from a non-bullous area of the back (Nikolsky's sign could be elicited nearby). The same basic histological picture was seen, with hyperkeratosis and well-marked acantholysis, but no bulla formation. The acantholytic cells appeared viable and had well-stained nuclei; and some of them showed the peripheral darkening of the cytoplasm observed in the previous biopsy.

A scraping from the base of an erosion produced by friction on the back was also stained and examined. The cells seen were those of the Malpighian layer, showing that cleavage had occurred within the stratum mucosum.

DISCUSSION

The histological picture in this case of bullous C.I.E. differs from most of the descriptions we have been able to find in the literature; histological reports deal almost always with skin from non-bullous lesions, and we have not seen any detailed description of a bullous lesion comparable with the one above.

In our case the histological picture bears a close resemblance to that of F.B.C.P. This dermatosis is generally recognized in Europe as a disease entity, but some North American authors maintain that it is only a variant of Darier's disease or of epidermolysis bullosa.

We feel that there is as much, or as little reason, clinically and histologically, to relate F.B.C.P. to C.I.E. (in its

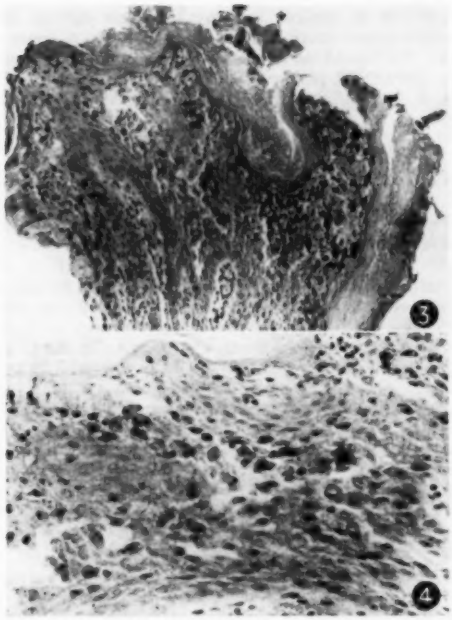

Fig. 3. Congenital ichthyosiform erythrodermia, showing hyperkeratosis and acantholysis with irregular intraepidermal bulla formation.
Fig. 4. Congenital ichthyosiform erythrodermia. Detail of Fig. 3 showing acantholysis. Note well-stained nuclei and distinct nucleoli in acantholytic cells.

bullous form) as to Darier's disease or epidermolysis bullosa, and that the question of the relationships of these bullous dermatoses deserves to be re-examined.

Familial benign chronic pemphigus (Gougerot-Hailey) was so named by the Haileys in 1939, but similar cases had been described earlier that year by Pels and Goodman [7] (as a bullous form of Darier's disease) and earlier still by Gougerot and Allée (1933). Zoon *et al.*[8] state that Zweig and Siemens had described such cases even before Gougerot, in 1920 and 1921.

F.B.C.P. is a stubborn, chronic, relapsing disease of familial distribution. The lesions are flaccid bullae, appearing on normal skin, which burst and leave crusted erosions. Nikolsky's sign may be positive on skin near the lesions. Healing leaves little trace. Sites of election are about the neck, axillae and groins. The lesions are usually localized, but large surfaces can be affected. The appearance, once the bullae have ruptured, is not striking, and many cases have been long unrecognized and treated as impetigo or eczema. The condition can be present at birth, but the first signs generally appear in the second or third decade.

The histological picture [9-12] is of an intra-epidermal bulla with dissociation of the elements of the stratum mucosum. The floor of the bulla is the stratum basale with, here and there, a few cells of the stratum mucosum still attached. The roof consists of a few layers of flattened parakeratotic cells to which, in places, some cells of the stratum mucosum still adhere. In the cavity lie freely the greater part of the cells of the stratum mucosum, isolated or in groups. The cavity is ill-defined and contains little fluid.

There is an extensive loss of the intercellular filaments of the stratum mucosum. The cells themselves are large, rounded and well stained, and show no real dyskeratosis. They seem to retain their vitality and ability to divide although they have lost their attachments, and mitotic figures can be seen in their nuclei. Keratinization begins deep in the epidermis in some of the detached cells; *grains* may be seen, but never *corps ronds*. There is little change in the dermis. The picture is similar to that of pemphigus vulgaris, but with less alteration of the cells of the stratum mucosum.

Darier's disease, of which F.B.C.P. has most frequently been said to be a variant, is also a familial disease which may present bullous lesions. It usually begins early in life and, unlike F.B.C.P., is permanent and progressive. Although the earliest lesions may be crops of small bullae,[13] there eventually develops a characteristic picture of follicular keratoses.

The histological picture is, in the opinion of the majority of European observers, and to our minds, quite different from that of F.B.C.P. Cleavage between the stratum basale and the stratum mucosum is commonly seen, but there is no generalized acantholysis. Indisputable evidence of dyskeratosis is given by the presence of *corps ronds* and *grains* in all the layers of the epidermis above the stratum basale. The dyskeratotic cells are smaller than normal and usually have pyknotic nuclei. The stratum basale shows characteristic tubular digitations down into the dermis (Fig. 5).

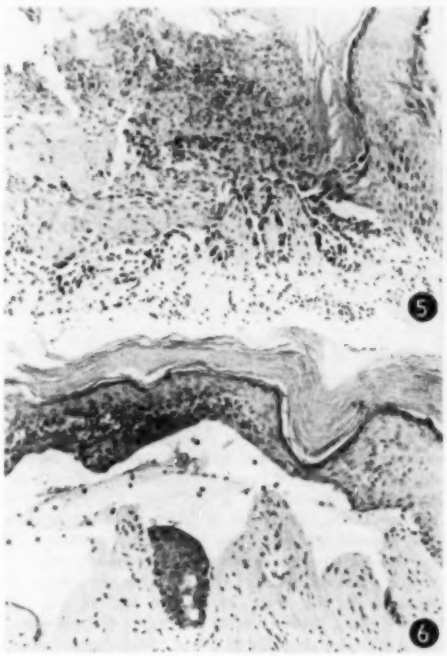

Fig. 5. Darier's disease. Note lacunae, tubular digitations of stratum basale and *corps ronds*.
Fig. 6. Epidermolysis bullosa (chronic porphyria). Subepidermal bulla with apices of two rete pegs attached to floor.

Epidermolysis bullosa is another familial disease characterized by bullae resulting from minor traumata. Although lesions may appear in the areas commonly affected in F.B.C.P., the sites of election are the hands and feet and some lesions are almost invariably found there. Porphyrinuria is commonly associated with epidermolysis bullosa (in South Africa at least [14]) and has not been reported as occurring with F.B.C.P. or C.I.E. The bulla of epidermolysis bullosa is formed by a detachment of the epidermis from the dermis, and the epidermis itself is not altered (Fig. 6). Degenerative changes in the superficial elastic tissue are described by some authors; others say that the dermis is unchanged.

Congenital ichthyosiform erythrodermia was fully described and named by Brocq in 1902 but similar cases had previously been noted by Vidal (1882) and others. It is a familial disease and lesions are present at birth or appear soon afterwards. Keratodermia, often generalized, is the essential feature; erythrodermia is not always present and Darier preferred to call the condition ichthyosiform hyperkeratosis.[15] Bullae are relatively commonly seen, especially in the earliest years. The distribution of the lesions distinguishes the condition from ichthyosis vulgaris and its variants; in C.I.E. the flexures are affected, in ichthyosis they are spared. Our case conforms so closely to Brocq's description that no further general detail is necessary.

In certain cases the nails are ridged and thickened and may grow 3 or 4 times as quickly as the normal; the hair may also grow quickly (Hyperépidermotrophie Generalisée, Vidal). The mucous membranes may be affected. There may be hyperhidrosis of palms and soles and the sebaceous secretion may be augmented on face and scalp. Associated malformations of the teeth, ears and palate have been described.[16]

The condition tends to improve with age; the erythrodermia disappears and bulla formation becomes less frequent.

The histological picture in non-bullous cases is of great hyperkeratosis, granulosis and acanthosis, with only minor changes of oedema and peri-vascular round cell infiltration in the dermis; sweat and sebaceous glands may be hypertrophied. In cases with bullae there is said to be marked acanthosis, great activity in the cells of the stratum basale and disorganization of the superficial elastic tissue. Bulla formation, according to Blum,[15] is a result of acantholysis. It has been suggested by Blum, Wile,[3] and others that this picture is reminiscent of epidermolysis bullosa.

Apart from the classical generalized form of C.I.E. there have been described, under a variety of names, a considerable number of cases which must be considered as variants or *formes frustes*. These cases are characterized by circumscribed, symmetrical patches of keratodermia with or without erythrodermia and bulla formation. The lesions may be fixed, progressive, recurrent or variable; in some cases lesions are present at birth, in others they appear later in life, even, rarely, as late as the fourth decade. Among these variants are found the palmar and plantar keratodermias (Unna-Thost, Maladie de Méléda) and Erythro- et Keratodermia Variabilis (Mendes da Costa).

Noordhoek [17] has recently studied a group of cases of *erythro- et keratodermia variabilis* occurring in successive generations of related families. His cases showed symmetrical lesions consisting of keratosis, erythrodermia or both. The lesions varied greatly in size, form and intensity. The histological picture was usually that of C.I.E. The sites of election for the lesions were often those of F.B.C.P.

CONCLUSIONS

The clinical points of resemblance between these 4 diseases are not striking. They all have bullous episodes, and the bullae may appear in the same regional distribution, but they have little else in common except resistance to all forms of treatment.

From the histological point of view it can be said at once that epidermolysis bullosa is unrelated to the other 3 dermatoses because the site of the cleavage producing the bulla is between epidermis and dermis. In C.I.E. and F.B.C.P. and Darier's disease the bulla is produced by a cleavage within the layers of the epidermis. F.B.C.P. and bullous C.I.E. are characterized by a profound acantholysis and dissociation of the cells of the stratum mucosum; and there is no definite evidence of dyskeratosis. In Darier's disease there is no generalized acantholysis; the cleavage occurs between the stratum mucosum and the stratum basale, there is a characteristic alteration of the stratum basale and striking evidence of dyskeratosis with *corps ronds* and *grains* (Table I).

The actual mechanisms of bulla formation, the cytological abnormalities on which it depends, are not known. It has been suggested, in the case of Darier's disease and C.I.E., that the bullae are simply manifestations of pyodermia because they are sometimes auto-inoculable.[12, 18] Any fissured epidermis is abnormally liable to infection, and some such cases do develop bullous impetigo; but the majority of observers do not agree that infection is a necessary prelude to bulla formation in all cases.

Our view is that there is insufficient evidence to suggest a relationship between any or all of these dermatoses. All may have bullae; but Brocq pointed out, 50 years ago, that congenital dermatoses are often characterized by bullae. The fact that the bullae of F.B.C.P., C.I.E. and Darier's disease are all produced by intra-epidermal cleavage need not imply that the causative process is identical in all 3 cases; and acantholysis and cleavage in this situation occur in other bullous dermatoses which are obviously unrelated to this group.

It seems more reasonable to accept the obvious clinical differences and ignore the minor and probably unimportant histological and clinical points of resemblance between these diseases, and to classify them as distinct and individual entities

SUMMARY

A case of bullous congenital ichthyosiform erythrodermia with an unusual histological picture, resembling that of familial benign chronic pemphigus, is described.

The relationship of these 2 dermatoses, and of other bullous dermatoses with which familial benign chronic pemphigus has been compared, is discussed.

TABLE I

	Familial Benign Chronic Pemphigus	*Congenital Ichthyosiform Erythrodermia*	*Darier's Disease*	*Epidermolysis Bullosa*
Age at onset	2nd or 3rd decade; but lesions may be present at birth or appear soon afterwards.	Lesions present at birth or appear soon afterwards; but may begin later in life.	Infancy or early childhood	3rd or 4th decade; but lesions may be present at birth or appear in early years.
Characteristic features	Flaccid bullae	Generalized, or symmetrical and localized, keratoses. Flaccid bullae are often seen especially in the generalized form and in the early years of life.	Follicular keratoses on body. Warty plaques on dorsa of hands and feet. Rarely, small vesicles or bullae.	Tense and flaccid bullae full of serum or blood-stained fluid.
Sites of election	Neck, axillae, groins; but spread may affect large areas. Palms and soles rarely, if ever, affected.	In discrete forms the large flexures and neck are often affected. Hyperkeratosis of palms and soles frequent.	Face, scalp, centre of chest and back, waist, large flexures. Often punctate keratoses of palms and soles.	Dorsa of hands and feet; forearms and legs; sometimes other friction points. Palms and soles sometimes affected.
Progress	Recurrent attacks. Skin normal between attacks; little or no scarring.	Usually permanent. A few cases of the discrete form have periods when skin is normal. Bullae come in crops; disappear without trace.	Permanent and often progressive. Temporary incomplete remissions in some cases.	Recurrent attacks. Some scars are generally visible between attacks.
Porphyrinuria	None	None	None	Frequent, but may be spasmodic. A single negative test is irrelevant.
Familial Incidence	Frequent	Frequent	Limited	Frequent
Treatment	Ineffective	Ineffective	Some cases appear to be temporarily improved by large doses of vitamin A.	Ineffective
Histological features	Acantholysis and dissociation of cells of stratum mucosum. *Grains* sometimes seen, but never *corps ronds*. No real dyskeratosis.	In bullous form acantholysis and great activity in stratum basale. Disorganization of superficial elastic tissue. No dyskeratosis. (In our case the picture was similar to that of F.B.C.P.).	Bulla formation results from cleavage between stratum basale and stratum mucosum. No generalized acantholysis. Stratum basale shows characteristic tubular digitations. Dyskeratosis, with *corps ronds* and *grains*, is a marked feature.	Bulla is formed by a simple detachment of epidermis from dermis. No inflammatory reaction. Dermis appears normal according to some authors; others have described degenerative changes in the elastic tissue. No dyskeratosis.

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ASSOCIATION NEWS : VERENIGINGSNUUS

CITATIONS READ AT THE OPENING CEREMONY OF THE SOUTH AFRICAN MEDICAL CONGRESS HELD IN JOHANNESBURG, ON 22 SEPTEMBER 1952

ASSOCIATION'S GOLD MEDAL FOR MERITORIOUS SERVICE

DR. J. S. DU TOIT

Dr. Jacobus Stephanus du Toit was born in Worcester, Cape, and was educated both there and at the Victoria College, Stellenbosch. He proceeded to Edinburgh in 1907 in order to study medicine. While there he was Treasurer of the South African Students' Club and also played rugby for Edinburgh University. He qualified to the degrees of Bachelor of Medicine and Bachelor of Surgery in 1912. In 1914 he received the degree of Doctor of Medicine and in the following year became a Fellow of the Royal College of Surgeons of Edinburgh.

While at Edinburgh he developed a desire to specialize in Ophthalmology and, after advanced study in this subject, he returned to Cape Town in 1916 where he has remained in practice. He soon became attached to the Department of Ophthalmology at the University of Cape Town and was joint head of the department for many years.

Dr. du Toit joined the British Medical Association while overseas and was later transferred to the Cape Western Branch of that Association. He served as Honorary Treasurer of the Branch for some years and was its President in 1929. He was associated with the move to form the Medical Association of South Africa (B.M.A.) and when this was brought into being in 1927 he was appointed Honorary Treasurer. He has occupied this position since that time and his long and faithful service is greatly appreciated by his colleagues. For the same long period he has served on the Executive Committee of the Federal Council and his membership of the Cape Western Branch Council extends over 35 years.

He has been a member of a number of committees both within and without the Association and his colleagues have always looked upon him as a wise counsellor. As a mark of their appreciation and high esteem they will themselves be honoured by his acceptance of the Association's Gold Medal for Meritorious Service.

DR. A. W. S. SICHEL

Dr. Alan William Stuart Sichel graduated to the degree of Bachelor of Arts in Classics at the South African College in 1906. He proceeded to the University of Edinburgh where he received with first class honours the degrees of Bachelor of Medicine and Bachelor of Surgery in 1912.

After a special mission to investigate trachoma on Christmas Island in the Indian Ocean, he saw service in France during World War I. Post-graduate study occupied the years immediately following the war and in 1920 he received from the University of Oxford the Diploma in Ophthalmology and from the Royal College of Physicians and Surgeons of London the Diploma in Ophthalmic Medicine and Surgery.

He commenced practice in Cape Town as an Ophthalmic Surgeon in 1921, and early interested himself in the affairs of the Medical Association of South Africa. He served the Cape Western Branch as Honorary Librarian for 14 years and has been a member of the Branch Council continuously since 1923. He was Organizing Secretary of the South African Medical Congress held in Cape Town in 1933 and was President of the Branch in 1941. He has been a member of the Federal Council of the Association since 1935, and a member of the Executive Committee since 1941.

He was joint lecturer in ophthalmology at the University of Cape Town from 1938 to 1949, and during World War II he served on the Consultative Council of the Director General of Medical Services as the Association's nominee.

In 1945 he was elected President of the Association, which office he held for six years. At the end of 1951 he was elected Chairman of the Federal Council and he holds that office at present.

In 1951 Dr. Sichel was particularly honoured, as was the Medical Association of South Africa, when he was elected to be the President of the British Medical Association to which our Association is affiliated. He thus had the unique distinction of being President of two Medical Associations at the same time.

The Medical Association of South Africa is proud to bestow on him its Gold Medal in appreciation of his outstanding service to the Association and the medical profession generally.

ASSOCIATION'S BRONZE MEDAL FOR MERITORIOUS SERVICE

DR. J. C. GIE

Dr. Johan Coenraad Gie was educated at the South African College and proceeded to London to study medicine in 1913. He served in the Royal Navy during World War I and in 1917 received the conjoint diploma of the Royal College of Surgeons of England and the Royal College of Physicians of London. In the following year he qualified to the degrees of Bachelor of Medicine and Bachelor of Surgery of the University of London.

On his return to South Africa he lectured in bacteriology at the University of Cape Town and then commenced practice at Sea Point where he has remained. He has served on the Honorary Staff of the Somerset Hospital, Cape Town, for many years. He early identified himself with the work of the Association in its Cape Western Branch and has served on a number of Committees. He was President of the Branch in 1940.

In earlier days a great deal of the Contract Practice work of the Association, outside the Southern Transvaal Branch area, was undertaken by a Committee of the Cape Western Branch, of which Dr. Gie was considered to be the mainspring. Later he saw the necessity to co-ordinate the Association's efforts in this branch of practice and, as a result of his representations, the present Central Committee for Contract Practice was brought into being. He has been a member of the Federal Council since 1943.

Dr. Gie has given hard work and enthusiasm to the Association and his colleagues are appreciative of his efforts on their behalf. Some of their appreciation is betokened by their request that he accept the Association's Bronze Medal for Meritorious Service.

DR. C. A. H. GREEN

With a distinguished record at school, Dr. Cyril Arnold Howell Green was awarded a Rhodes Scholarship and proceeded to Oxford to study medicine, following the footsteps of his father. In 1924 he qualified to the degrees of Bachelor of Medicine and Bachelor of Surgery of Oxford and at the same time received the conjoint diploma of the Royal College of Surgeons of England and the Royal College of Physicians of London. He returned to South Africa to practice in the same year. Most of his life since that time has been spent in Johannesburg and on the Reef and he has a record of devoted service to his many patients.

Dr. Green has been a member of the Ethical Committee of the Southern Transvaal Branch for many years, as also both a member and Chairman of the Branch Contract Committee. From 1942 to 1945 he was Honorary Secretary of the Branch and was its President in 1948. He was elected to membership of the Federal Council in 1943. When the Central Committee for Contract Practice was set up he

became its first Chairman and his work in this sphere will be particularly remembered.

Dr. Green has served the Association faithfully and well and his colleagues are glad of the opportunity to pay him tribute by means of the Association's Bronze Medal.

DR. SHADICK HIGGINS

Dr. Tom Shadick Higgins had a distinguished academic career in London, receiving the degree of Bachelor of Science with Honours in 1905, the conjoint diploma of the Royal College of Surgeons of England and the Royal College of Physicians of London in 1907 and the degrees of Bachelor of Medicine and Bachelor of Surgery of the University of London with honours in the same year. In the following year he received the Diploma in Public Health of the University of Cambridge and in 1911 the degree of Doctor of Medicine in State Medicine of the University of London.

After a period of service as Medical Officer of Health of the Metropolitan Borough of St. Pancras, London, he came to South Africa to be the Medical Officer of Health of the City of Cape Town. He was, at the same time, appointed as Professor of Public Health at the University of Cape Town, and he occupied these positions until his retirement in 1944.

Soon after his arrival in Cape Town, Dr. Shadick Higgins identified himself whole-heartedly with the work of the Association and he has been an active member of many committees. He has been a member of the Council of the Cape Western Branch for over 25 years and at various times has been Chairman of the Branch Ethical Committee and the Parliamentary Committee. He was President of the Cape Western Branch in 1930 and has served on the Federal Council since 1938. From 1943 to 1948 Dr. Shadick Higgins was a member of the South African Medical and Dental Council and of its Education Committee. For four years he represented that Council on the South African Nursing Council. In addition he has served on various Government and Provincial Commissions.

In all these undertakings Dr. Shadick Higgins has shown a knowledge, vision and judgment which have made his guiding influence of the greatest value to the Association and, indeed, to the medical profession. In appreciation of these qualities his colleagues wish to bestow on him the Association's Bronze Medal for Meritorious Service.

CITATIONS WERE READ ON 22 SEPTEMBER 1952 AT THE OPENING CEREMONY OF THE SOUTH AFRICAN MEDICAL CONGRESS HELD IN JOHANNESBURG, WHEN CERTIFICATES OF HONORARY MEMBERSHIP WERE PRESENTED TO THE FOLLOWING SCIENTISTS:

DR. R. A. ALEXANDER

Raymond Albert Alexander was educated at Jeppe High School, Johannesburg, Potchefstroom School of Agriculture and, after World War I, at the Transvaal University College. In 1922 he was awarded the Bachelor of Science (Agriculture) degree and in 1925 the Bachelor of Veterinary Science degree (with honours). In 1935 he was awarded the Doctor of Veterinary Science (South Africa) degree, the subject of his thesis being 'Studies on the Neurotropic Virus of Horse-Sickness'. It was from this work that the present method of immunization against horse-sickness was developed.

In 1926 he was appointed a Veterinary Research Officer at Onderstepoort in the Division of Veterinary Education and Research of the Department of Agriculture. He moved for a period to the Alberton Research Laboratory and in 1928 he returned to Onderstepoort and joined the Section of Virus Diseases and Protozoology as Professional Assistant to the Director. In 1929 he was appointed Lecturer in Infectious Diseases in the Faculty of Veterinary Science, Onderstepoort. In 1931 the Empire Marketing Board of Great Britain established three research fellowships and he was appointed from South Africa.

Thereafter followed a period of research in Cambridge, visits to major research institutes in European countries and continued research work into virus diseases at Onderstepoort as a Research Fellow. In 1938 he was appointed Senior Research Officer at Onderstepoort in charge of all research work into diseases carried by ultra-visible viruses.

In the succeeding 10 years he visited America and Canada, proceeded to Egypt to organize the control of an extensive outbreak of horse-sickness in Egypt and the Middle East, and visited England, Holland and Switzerland in connection with the control of foot-and-mouth disease in cattle.

In 1948 he was appointed Sub-Director, in 1949 was made Deputy Director and in 1950 succeeded to the post of Director of Veterinary Services.

He has contributed approximately 70 technical articles to various scientific journals, chiefly on subjects connected with virus diseases. His research work on virus diseases has been of considerable value to the medical profession and he has revealed himself as a research worker and scientist of outstanding ability and merit.

We are honoured to elect him an Honorary Member of the Medical Association of South Africa.

DR. GILLES DE KOCK

Dr. Gilles (van de Wall) de Kock obtained his diploma as a Member of the Royal College of Veterinary Surgeons of London in 1913 and in the following year, as Research Officer, commenced his long association with the Veterinary Research Institute at Onderstepoort and with the Veterinary Faculty of the University of Pretoria. This association was interrupted for only one year when he accepted the Chair of Anatomy at Stellenbosch. In 1920 he returned to Onderstepoort as Professor of Anatomy and Senior Research Officer under the late Sir Arnold Theiler. In 1922 he received a Doctorate in Veterinary Medicine in Switzerland and in 1928 he was awarded the Doctor of Science degree by Witwatersrand University.

During his distinguished research career he applied himself in the first place to the study of blood diseases and the effects of splenectomy. Later, stimulated by his association with men like Ludwig Aschoff, he developed the keen interest in histopathology which remains with him to-day. He has also been actively associated with the successful campaign against the tsetse fly in this country, and in the control of other communicable diseases of animals.

He rose from the position of Research Officer to that of Deputy Director of the Veterinary Institute in 1931 and in 1948 he became Director of Veterinary Services. He was also Dean of the Veterinary Faculty of his University.

The Senior Captain Scott Medal of the South African Biological Society was awarded to him in 1934, and the South African Medal and Grant of the South African Association for the Advancement of Science in 1949, the year of his presidency of that Association.

Dr. de Kock has, throughout his career, worked for the advancement of science in South Africa, not only in his own field but also in particular the field of human medicine, which shares so many of its problems with veterinary medicine. His enthusiasm has been largely responsible for the promotion of a closer co-operation between doctors and veterinarians.

Since his retirement in 1949 Dr. de Kock has been active in his co-operation with medical research workers, and it is fitting that he be admitted to Honorary Membership of the Medical Association of South Africa by his medical colleagues who appreciate the value of his work.

DR. BOTHA DE MEILLON

Dr. Botha De Meillon started on his career of research at the South African Institute for Medical Research in 1926. He had just completed the Bachelor of Science course at the University of the Witwatersrand, being awarded first class honours in Zoology. Later he received the degrees of Doctor of Science and Doctor of Philosophy. He is also a Fellow of the Royal Entomological Society.

He had already revealed a keen interest in field studies and his extensive contributions to our knowledge of the human parasites and biting insects of Southern Africa were based on those early interests. These studies soon brought world-wide recognition and he has for many years been

acknowledged to be the leading entomologist in Africa. He has approximately 100 original contributions to scientific journals to his credit. Of particular value in the medical field are the studies he has completed and those on which he is still engaged concerning the vectors of malaria, yellow fever and bilharziasis.

DR. P. J. DU TOIT

Petrus Johann du Toit was educated at the Boys' High School, Wellington, and Victoria College, Stellenbosch. In 1907 he was awarded the Bachelor of Arts degree with honours and the Queen Victoria Scholarship for further study overseas.

It was the late Dr. Robert Broom who aroused his interest in Zoology and this became the main subject in his degree course. His studies overseas were primarily in Germany and Switzerland. In 1912 he obtained the degree of Doctor of Philosophy with honours in Zoology, Comparative Anatomy and Botany at the University of Zürich.

At the request of the Government of South Africa and with the aid of a Government scholarship, he proceeded to Berlin where he took up the study of Veterinary Science, obtaining the Diploma of Veterinary Surgeon in 1915 and the degree of Doctor of Veterinary Medicine with honours in 1916.

Following a period of further research he returned to South Africa in 1919 and was appointed Senior Veterinary Officer in the Department of Agriculture under Sir Arnold Theiler. A year later he was appointed Sub-Director of Veterinary Education and Research and Professor of Infectious Diseases in the Faculty of Veterinary Science in the University of South Africa.

In 1927 he succeeded Sir Arnold Theiler as Director of Veterinary Services and was appointed Professor of Tropical Diseases and Dean of the Faculty of Veterinary Services. On retiring from these posts in 1948 he was appointed Deputy President of the South African Council for Scientific and Industrial Research, and in 1950 he was appointed President.

He has filled several other posts with distinction which included President of Section 'M' of the British Association for the Advancement of Science, Bristol, England, in 1930.

Many public and academic honours have been bestowed upon this distinguished scientist, including honorary doctorates of the Universities of Stellenbosch, Cape Town, Utrecht and Witwatersrand; Honorary Doctor of Laws of Glasgow and Honorary Associate of the Royal College of Veterinary Surgeons, London.

Amongst his numerous publications was 'Tropical Diseases of Animals' which has a very intimate bearing on human diseases. He was awarded the Bernard Nocht Medal for Tropical Medicine, Hamburg, Germany, in 1938. This award was made annually for outstanding work in tropical diseases and he is the only non-medical recipient of this award.

Medical research is the backbone of medical progress. In South Africa the Council for Scientific and Industrial Research is charged *inter alia* with the development of medical research. A specially constituted committee, styled the Medical and Dental Research Committee, acts as an advisory committee to the Council. Dr. du Toit is not only President of the Council but is also a member of the Advisory Committee. He furthermore serves on certain very specialized research sub-committees, such as the sub-committee controlling the Nutrition Unit of the University of the Witwatersrand.

Thus this most eminent scientist has been and is very closely linked with the medical profession by his achievements and his work, and we are proud to be able to elect him an Honorary Member of the Medical Association of South Africa.

DR. F. W. FOX

Dr. Francis William Fox became an Associate of the Royal College of Science in 1917, in which year he was awarded the Bachelor of Science degree of London University with honours in Chemistry. His interest from the beginning was in the chemistry of nutrition. In 1917 he was engaged in analytical work for the Food Committee of the Royal Society set up by the military authorities during World War I. In 1919 he held the Lindley Studentship in Physiological Chemistry and his research work in this subject was rewarded by London University in the following year with the Master of Science degree. From 1920 to 1924 he held a Beit Memorial Fellowship for Medical Research at St. George's Hospital Medical School.

He was appointed to the post of Biochemist at the South African Institute for Medical Research in 1925. The following year he was awarded the Doctor of Science degree by London University for his work on the composition of human bile and its bearing on cholesterol metabolism. He was responsible for many publications on biochemistry, particularly in its relation to human nutrition. Latterly he has gone far beyond the mere chemical aspects of our malnutrition problem and has delved deeply also into the educational, economic and agricultural aspects.

Dr. Fox is a member of the South African Chemical Institute and the South African Association for the Advancement of Science, being President of the former in 1931 and of Section 'B' of the latter in 1939. He was a Government nominee on the National Food Council from 1943 to 1944 and was also nominated by the Government to the National Nutrition Council, the Soil Conservation Board and the South African National Food and Agriculture Organization Liaison Committee.

DR. H. ZWARENSTEIN

Dr. H. Zwarenstein received his first academic qualification of Bachelor of Arts in 1919. Already he had established an association with medicine, in that the subjects for his degree included Anatomy and Physiology. In the following year he obtained his Master of Arts degree in Physiology, with distinction. In the next few years he obtained the Bachelor of Science and the Doctor of Philosophy degrees of Manchester University as well as the Master of Science degree of the University of Cape Town, and in 1939 he was awarded a Doctorate of Science by Manchester University.

His close association with medicine has continued throughout the years. Since 1920 he has held academic appointments in Physiology in the Universities of Cape Town and Witwatersrand, during all of which time he has been concerned with teaching of medical students. He is at present senior lecturer in Physiology at the University of Cape Town. Both his students and colleagues will remember him not only as a stimulating teacher, but also for his distinguished research work in the field of endocrinology and biochemistry. Out of his work on the pituitary gland arose one of his most outstanding contributions to medicine, the Xenopus test for pregnancy known throughout most of the world as the Zwarenstein-Shapiro test. In 1936 Dr. Zwarenstein was elected to a fellowship of the Royal Society of South Africa.

OFFICIAL ANNOUNCEMENT : AMPTELIKE AANKONDIGING

VACANCY FOR EDITOR

Applications are invited from registered medical practitioners for the post of Editor of the South African Medical Journal and the South African Journal of Clinical Science. The salary scale is £1,500 × 50—£2,000 plus cost-of-living allowance at Public Service rates. The post is full-time and the successful

VAKATURE VIR REDAKTEUR

Aansoeke van geregistreerde geneeshere vir die vakante betrekking van Redakteur van die Suid-Afrikaanse Tydskrif vir Geneeskunde en die Suid-Afrikaanse Tydskrif vir Kliniese Wetenskap word ingewag. Die salarisskaal is £1,500 × 50—£2,000 plus duurtetoeslag volgens Staatsdienstarief. Dit is 'n

applicant will be required to work at the Association's Head Office in Cape Town.

Applicants should state their experience and whether they are fully bilingual.

Applications should be addressed to the undersigned and should reach him before 31 January 1953.

A. H. Tonkin,
Secretary.

Medical House
35 Wale Street,
Cape Town.
24 October 1952.

voltydse betrekking en die aangestelde persoon sal verwag word om by die Vereniging se Hoofkantoor in Kaapstad werksaam te wees.

Applikante moet vermeld watter ondervinding hulle het en of hulle volkome tweetalig is.

Aansoeke moet gerig word aan die ondergetekende en moet hom voor 31 Januarie 1953 bereik.

A. H. Tonkin,
Sekretaris.

Mediese Huis,
Waalstraat 35,
Kaapstad.
24 Oktober 1952.

PASSING EVENTS

Dr. L. I. Braun, the President of the Johannesburg Medical Congress, was the recipient of an honorary LL.D. degree during Congress Week in September 1952, from the University

Dr. L. I. Braun

of the Witwatersrand. Other members of the Association thus honoured were: Dr. A. W. S. Sichel, Chairman of the Federal Council of the Medical Association of South Africa, The Honourable The Minister of Health, Dr. Karl Bremer, M.P. (*in absentia*) and Dr. H. A. Moffat (*in absentia*).

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Dr. I. Simson Hall will be in Port Elizabeth until 25 December, when he will deliver a lecture. Dr. Simson Hall is a senior member on the Ear, Nose and Throat Staff of the Edinburgh Royal Infirmary.

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Dr. Werner Weinberg of Johannesburg has been invited to act as a Contributing Editor to *International Abstracts*, a new section which will be inaugurated early in 1953 in the *Fertility and Sterility Journal* published by the International Fertility Association.

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Dr. C. R. Woolf of Cape Town has passed the examinations for admission to Membership of the Royal College of Physicians (London).

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The Office of the Workmen's Compensation Commissioner, P.O. Box 955, Pretoria, has issued a booklet entitled *Occupational Health and Safety: A Course for Industrial Apprentices.*

This booklet has been prepared by Dr. Gerald Machanik, Medical Officer in the Office of the Workmen's Compensation Commissioner.

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A selected list of the latest accessions to the Medical Library, Mowbray, for the period July—September 1952 is now ready. Members of the Medical Association may receive copies upon application to the Medical Library, Medical School, Mowbray, C.P.

REVIEWS OF BOOKS

OCULOROTARY MUSCLES

The Oculorotary Muscles. By Richard G. Scobee, B.A., M.D., F.A.C.S. Second Edition. (Pp. 512, with 159 illustrations. 93s. 6d.) St. Louis: The C. V. Mosby Company. 1952.

Contents: Section I—The Background of Ocular Deviations. 1. Anatomy. 2. Synergists, Antagonists, Yoke Muscles. 3. Laws of Innervation—Primary and Secondary Positions of Gaze. 4. Binocular Factors. 5. Physiology and Mechanics of the Oculorotary Muscles. 6. Neurophysiology of the Oculorotary Mechanism. *Section II—Latent Deviations.* 7. The Position of Rest. 8. Orthophoria. 9. Heterophoria. 10. Esophoria. 11. Exophoria. 12. Hyperphoria. 13. Cyclophoria. *Section III—Manifest Deviations.* 14. Heterotropia. 15. Concomitant Heterotropia. 16. Noncomitant Heterotropia. *Section IV—Making the Diagnosis.* 17. The History. 18. Visual Acuity. 19. The Angle Kappa. 20. Measuring the Deviation. 21. Ductions. 22. Versions—Primary and Secondary Deviations. 23. Vergences. 24. Diplopia Fields. 25. Other Diagnostic Points. 26. The Diagnosis of Hypertropia. 27. Analysis of the Case. *Section V—Treatment of the Deviation.* 28. The Nonsurgical Treatment of Heterotropia. 29. The Surgical Treatment of Heterotropia. 30. Reasons for Failure in Tropia Surgery. Appendix. Specific Testing Technique for the Maddox Rod.

In describing this book as 'a simple but accurate and workable approach to the subject of the ocular muscles and their dysfunction' the author has not done full justice to himself. He has done considerable research in this subject and has carefully analysed the literature, debunking much general loose thinking. He has crammed 502 pages of text full of the most detailed and critical information about the normal anatomy and physiology of the oculorotary muscles; the latent and manifest deviations, their diagnosis and treatment. Because of this amount of detail the first part of the book is

a little difficult, though never dull. Throughout the exposition is clear and definite.

Certain chapters are particularly worth noting, such as the amount of surgery to be performed. It is emphasized that there is no rule of thumb to decide exactly how many millimetres of surgery should be done before operation. About the only thing which can be decided in advance is the type of operation to employ. The final decision must be made on the operating table.

The factors to be considered are detailed and discussed. Two aspects of the subject (correctly in the reviewer's opinion) are not described in great detail—orthoptics (which can be found elsewhere); and surgical techniques. He only describes the techniques used by himself and found satisfactory. Here the reader is advised to use whichever technique he prefers.

The book concludes with an analysis of the causes of failure in tropia surgery which is worthy of study. Already in its second edition, this book should find its way to the ophthalmologist's bookshelf.

General Practitioner's Refresher Course

Refresher Course for General Practitioners. Edited by the Editor, *British Medical Journal*. (Pp. 486 + 10, with illustrations. 25s.) London: British Medical Association.

Contents: 1. The Acute Throat. 2. The Acute Ear. 3. Failed Forceps. 4. Use and Abuse of Vitamins. 5. Management of the Premature Baby. 6. Fracture of the Clavicle. 7. Management of Acute Pneumonias in Adults. 8. Chilblains. 9. Management of Diabetes Mellitus. 10. Acute Dyspepsia. 11. Analgesia in Childbirth. 12. Early Diagnosis of Bronchial Carcinoma. 13. Diagnosis of Early Phthisis. 14. Measles. 15. The Influenzal Diseases. 16. Bronchopneumonia in Infancy. 17. Acute and Chronic Sinusitis. 18. Early Diagnosis of Tuberculous Meningitis. 19. Fracture of the Carpal Scaphoid. 20. Acute Abdominal Disease. 21. Post-Partum Haemorrhage. 22. Causes and Treatment of Eczema. 23. Headache. 24. Menorrhagia. 25. Modern Views on Uraemia and Dehydration. 26. Hypertension. 27. Infant Feeding. 28. Congenital Heart Disease. 29. Management of Asthma. 30. Delay in the Second Stage of Labour. 31. Diagnosis and Treatment of Pernicious Anaemia. 32. Osteoarthritis. 33. Conjunctivitis and Iridocyclitis. 34. Cardiac Neuroses. 35. Management of Pulmonary Tuberculosis. 36. The Problem of Psoriasis. 37. Carcinoma of the Stomach. 38. The Failing Heart. 39. Sulphonamide Therapy. 40. Penicillin. 41. Streptomycin. 42. The Newer Antibiotics. 43. Poliomyelitis. 44. Pink Disease. 45. Electro-cardiography. 46. Treatment of Ulcerative Colitis. 47. Migraine. 48. Management of the Epileptic. 49. Enuresis. 50. Addison's Disease. 51. Menstrual Disorders and the Menopause. 52. Treatment of Varicose Veins. 53. Management of Thyrotoxicosis. 54. Treatment of Lumbar Disc Lesions. 55. Bronchiectasis. Index.

This volume gathers together into convenient compass articles which appeared in the *British Medical Journal* from October 1949 to December 1950. These articles were specially commissioned as a refresher course for general practitioners, at whose request they have been put together in this accessible and handy fashion. It is certain that this volume will fulfil a long-felt and much needed requirement, because, in the words of Dr. Hugh Clegg, the Editor of the *British Medical Journal*: 'At no time in the practice of medicine has the need been so urgent for post-graduate education of one sort and another. The medical scene changes so rapidly and so many remedies are being put at the disposal of the doctor and his patient that a current and practical review of progress is essential.'

This volume also serves the valuable purpose of keeping specialists informed about developments in other fields than their own, and our distinguished contemporary *Journal* is to be congratulated on its intelligent anticipation of the needs of the profession.

Taken in conjunction with the first and second series of *Any Questions?*, published in the last 2 years, the present volume provides stimulating, practical and instructive information.

Readers can look forward to further collections of articles featuring as the *Refresher Course for General Practitioners* in the pages of the *British Medical Journal*.

Ankylosing Spondylitis

La Spondylarthrite Ankylosante. By J. Forestier, F. Jacqueline and J. Rotes-Querol. (Pp. 330, with 143 figures. 2,650 Fr.). Paris: Masson et Cie. 1951.

This work on ankylosing spondylitis relates experiences and observations on over 400 personal cases. The incidence of the disease is reviewed and it is reported as a relatively frequent malady, affecting males 10 times more commonly than females.

The insidious and intermittent opening phase, marked by root pains, involvement of peripheral joints, iritis, diffuse pains and radiological states in the sacro-iliac joints, is outlined briefly. Thereafter, the established phase of persisting lumbar pains, contraction and atrophy of lumbar muscles, thoracic pains and diminished costo-vertebral movement follows. Further details relating to the spine, the invalidism from peripheral joint involvement, the sacro-iliac ankylosis, the systemic symptoms and signs are outlined clearly. The same is true of the third or advanced phase, with ankylosis of the greater part of the spine and peripheral ankyloses. After this brief preliminary introduction, which gives the over-all picture, there are detailed descriptions of spinal and juxta-spinal features, clinical and radiological. Referred pain and the state of the peripheral joints are very fully described, as are the ocular manifestations which are so common as to constitute symptoms rather than complications.

Laboratory investigations, further evolution of the disease and diagnostic problems receive full attention. Thereafter follows a study of the etiology and treatment. The bibliography of 221 references concludes the work.

This appears to be an authoritative publication of the subject, of considerable interest, and written in French in a simple and concise manner.

Pregnancy Toxaemias

The Toxemias of Pregnancy. By W. J. Dieckmann, S.B., M.D. (Pp. 710, with 85 illustrations. £6 3s. 3d.) Second Edition. St. Louis: C. V. Mosby Co. 1952.

Contents: *Section I—History, Classification, Incidence and Pathology of the Toxemias of Pregnancy.* 1. The Toxemias of Pregnancy. 2. Incidence of Eclampsia and Other Toxemias of Pregnancy. 3. The Pathology of Eclampsia.

Section II—Normal and Abnormal Physiology. 4. Physicochemical Determinations. 5. The Blood Pressure. 6. Renal Physiology. 7. Anatomy, Physiology and Pathology of the Liver. 8. The Ocular System. 9. Anatomy, Physiology and Pathology of the Placenta. 10. The Endocrine Glands. 11. Estrogens and Gonadotropins. 12. Edema in Preeclampsia and Eclampsia.

Section III—The Etiology of Eclampsia. 13. Factors Influencing Eclampsia in Pregnant Animals. 14. Factors Influencing Eclampsia in Pregnant Women. 15. Constitutional and Environmental Factors.

Section IV—Clinical Aspects of the Toxemias of Pregnancy. 16. Signs and Symptoms. 17. Hypertensive Disease in Pregnancy. 18. Acute Diseases of the Kidney. 19. Chronic Diseases of the Kidney. 20. Types of Eclampsia. 21. Preeclampsia and Eclampsia. 22. Complications of Toxemia.

Section V—The Treatment of the Toxemias of Pregnancy. 23. Historical Résumé of the Treatment of Eclampsia. 24. Pharmacodynamics. 25. Procedures and Methods of Treatment. 26. Termination of the Pregnancy. 27. The Obstetric Treatment of Eclampsia. 28. The Obstetric Treatment of Non-convulsive Toxemia of Pregnancy.

Section VI—Maternal and Fetal Prognosis and Prenatal Care. 29. Maternal Mortality. 30. Fetal Mortality. 31. Maternal and Fetal Sequelae. 32. Prenatal Care. Monographs.

To review this monograph, one must compare it with a large cake, containing a wealth of the best ingredients, baked at the right temperature, beautifully decorated and extremely wholesome, if not a bit heavy.

The *Toxemias of Pregnancy* is a magnificent monument to a pioneer who has devoted 28 years of his life's work to an investigation into the cause of so intriguing yet so baffling a condition. Almost all the new facts (from 1940 to present day) from the avalanche of world literature pertaining to this subject, are presented, not only for the obstetrician, but also for the worker untrained in midwifery.

Divided into 6 sections with 32 chapters, this book embraces not only the detailed anatomy and physiology of the various systems, but also every aspect of the main symptoms and signs related to the toxaemias and allied conditions, with dozens of case histories to illustrate the text.

The amount of work entailed in compiling this manuscript can be judged from, e.g., Chapter IV, meant (one is sure) more for the research worker, where 80 pages have been devoted to physico-chemical analysis, and where 253 authors are quoted.

The pathology of eclampsia and allied conditions with the most recent work on placental infarction and the hormonal aspects of the toxaemias and diabetes are minutely examined and beautifully illustrated.

Widely useful in everyday practice

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Diekmann's own contribution is to be found throughout this book, particularly in the field of sodium and water retention. Reduction in sodium, increase of water excretion, promotion of haemodilution, and relief of arteriolar spasm after delivery are his keynotes in the treatment of eclamptics at the Chicago Lying-In Hospital.

The proper use of intravenous hypertonic glucose solutions (20 to 50%) and their ability to control fits with small amounts of intramuscular magnesium sulphate are stressed as being one of the outstanding accomplishments of modern obstetrics. Maternal and foetal mortality is fully discussed, with a view to constantly improving methods by which the death rate can be reduced. Hence a very minute analysis of various obstetric treatments is outlined with emphasis on the reduction of the caesarean section rate and increase in low forceps (even with Duhrrsen's incisions of the cervix) under non-inhalation anaesthesia, which has so improved infant mortality, in premature babies from toxaemic mothers.

Much attention is paid to the after-care of this infant, the mortality of which has been as high as 13% when delivered by caesarean section, mainly because of prematurity.

An excellent chapter on future pregnancies and prognosis of the mother with previous toxaemia, and its legal implications, concludes this book.

Its high price and large size should not detract all practising obstetricians and research workers from making this volume a 'must' for their library shelves.

GYNAECOLOGY

Handbook of Gynaecological Diagnosis. Walter Neuweiler, M.D. (Pp. 448 + xiii, with 406 illustrations. 80s.) London: William Heinemann Medical Books Ltd.

Contents: 1. Case History of Gynaecological Disorders. 2. Examination. 3. Examination by Instruments. 4. Special Methods of Examination. 5. Diseases of the External Genitals. 6. Diseases of the Vagina. 7. Diseases of the Uterus. 8. Diseases of the Adnexa. 9. Diseases of the Abdominal Wall. 10. Ectopic Pregnancy. 11. Diseases of the Pelvic Cellular Tissue. 12. Endometriosis. 13. Discharge. 14. Sterility. 15. Low Backache.

This book (first published in 1952 and intended primarily for the general practitioner and student) discusses very fully the whole field of gynaecology and is well illustrated throughout.

The 95 pages devoted to the clinical examination will well repay careful study. The description of the colposcope and its clinical applications are of special interest in view of the recent visit to Cape Town of Prof. Hinselmann whose work he quotes at length.

The 'special part' of the book devoted to the symptoms and diagnosis of individual diseases makes rather heavy reading and in parts tends to be confusing.

The author's explanation of vaginal bleeding in newly-born female children as the consequence of a 'decrease of blood coagulation caused by hypoprothrombinaemia' will not be generally accepted—nor his insistence on a rectal temperature for a basal temperature chart, while his charts on page 283 fail to show the fall of temperature which accompanies the onset of menstruation.

While one might object to his classifying pregnancy as a cause for physiological sterility (p. 422) and Rhesus incompatibility as a cause for secondary sterility, this book can be strongly recommended for the student and particularly so for those intending to specialize in gynaecology.

TROPICAL DERMATOLOGY

Handbook of Tropical Dermatology and Medical Mycology. Vol. I. Edited by R. D. G. Ph. Simons. (Pp. 845 + x with 587 illustrations. £5.) Amsterdam: Elsevier Publishing Company, Ltd.

Contents: *List of Contributors. Preface.*
General Survey Dermatology Among Children. Pigmentations, Depigmentations. 1. General Survey. 2. Some Aspects Peculiar to the Practice of Dermatology among Children in the Tropics. 3. Pigmentary Disorders. 4. Depigmentation (Achromia).
Diseases Due to Protozoa Spirochaetes and Related Conditions. 5. Dermatological Syphilis in Non-Whites, particularly in the Negro. 6. Bejel. 7. Yaws. 8. Pinta or Carate. 9. Goundou. 10. Rat-bite Fever. 11. Cutaneous Leishmaniasis. 12. American Leishmaniasis or Leishmaniasis Muco-cutanea. 13. South American Trypanosomiasis (Chagas' Disease). 14. Cutaneous Amoebiasis.
Diseases Due to Bacteria. 15. Ulceration in the Tropics. 16. Tropicaloid Ulcer or Mycetoid Desert Sore. 17. Noma de Madagascar (Oral Homamadana). 18. Cutaneous Diphtheria. 19. Leprosy. 20. Cutaneous Tuberculosis and Sarcoidosis in the American Negro and in Inhabitants of Tropical Countries. 21. Cutaneous Plague. 22. Scleroma Respiratorium (Rhinoscleroma). 23. Granuloma Venerum (Donovanosis).
Diseases Due to Cocci. 24. Pyoderma. 25. Pyosis Mansoni (Monkey Pox or Impetigo Bullosa). 26. Tropical Pyomyositis (Bung Pagga).
Diseases Due to Rickettsiae and Viruses. 27. The Rickettsioses. 28. Exanthemata in the Dengue Group. 29. Bartonellosis. 30. Variola (Smallpox). 31. Lymphopathia Venerea.
The Viliaria Group of Dermatoses and Tropical Acne. 32. The Miliaria Group of Dermatoses (including Prickly Heat). 33. Tropical Acne. Index.

A long-standing need for a textbook on tropical diseases of the skin has been met by this volume.

Dr. Simons of Leyden University is well known in the dermatological world for his experience in tropical medicine. In addition to writing several of the main chapters himself, he has allotted to 40 individual specialists that particular subdivision of tropical dermatology on which he is an expert. The result of this arrangement has led to a really excellent reference book on tropical skin diseases.

It is well known that the everyday skin diseases of temperate climates, e.g. impetigo, acne, psoriasis, seborrhoeic dermatitis, fungus diseases, etc. are greatly modified by tropical conditions. Racial, nutritional and parasitical factors as well as sunlight, heat and moisture will completely alter the clinical aspects of most diseases.

There are many skin diseases that are more or less confined to the tropics. A full and detailed survey of these diseases is presented. Amongst the many outstanding chapters are those dealing with the various aspects of leishmaniasis and leprosy. The different and varied clinical pictures in which leishmaniasis can present itself is amazing.

The Editor rightly makes a strong plea for a simplication of the current nomenclature used in tropical medicine.

The illustrations are ample both in quality and quantity. Altogether this is an instructive and educative book which every dermatologist and physician interested in tropical diseases should possess.

CORRESPONDENCE

WORLD MEDICAL CONGRESS FOR THE STUDY OF PRESENT LIVING CONDITIONS

To the Editor: The President and the International Secretariat of the World Medical Congress for the Study of Present Living Conditions, which was to have been held in Montecatini, Italy, from 16—18 October 1952, regret to announce that the Italian Government has vetoed this Congress. The prohibition was issued, without any explanation, at the last moment, when preparations were already complete and when all the reports had already been written.

It is impossible to believe that the Italian Government should have any objection concerning the members of the Congress, since these include many famous personalities in the medical world of 34 countries. Hundreds of doctors, in all parts of the world, without exception, have manifested their interest and have held many preparatory meetings to discuss beforehand the various points of the programme. The scientific standard of the reports, contributed by eminent personalities, is very high.

All those who have taken part in the preparatory work, first of all the reporters, know that the Congress has no other aim than that of a medical international meeting. They can testify that they have always had complete freedom for the choice of the contents, the material and the presentation of their contribution to the Congress.

It is obvious that this prohibition is a blow to the liberty of exchanging scientific information between the different countries of the world. It will cause surprise and dismay among all scientific workers.

For this reason and because of the universal interest in the Congress, the International Secretariat feels itself bound to proceed with the organization of the Congress, which will be held as soon as possible, in any case not later than April 1953. Further news about the new arrangements will be issued later.

The International Secretariat urges all doctors to support the World Medical Congress for the Study of the Present Living Conditions and to proceed with the preparatory work in order to secure its success.

Pietro Verga,
President.
Claudio Massenti,
For the International Secretariat.

Congres Mondial des Medecins,
Pour l'Etude des Conditions Actuelles de Vie,
Corso Triesté, 65,
Rome.
7 October 1952.

An Unusual Response

To the Editor: In a series of well over 100 intra-sinal instillations of penicillin *cum* hyaluronidase, many repeatedly in the same subject, some in known allergic individuals, the writer had not encountered any sensitivity reactions until the following episode.

During the management of bilateral suppurative sphenoiditis in a 45-year-old female, 50,000 units of pencillin and 250 Benger units of hyaluronidase in 1 c.c. of sterile normal saline were introduced into each sphenoid sinus in the Proetz position following catheterization of the natural openings. Within 5 minutes of removal of the catheter and with the patient still supine waves of intense itching commenced in both palms, both external auditory canals and the external genitalia. Twenty minutes after the onset the palms and the external ears became intensely pink, and the patient was given one 25 mg. antihistaminic tablet orally. The redness of the palms now began to deepen, the distal 2 phalanges of all the fingers as well as the dorsum of both hands becoming intensely cyanotic. Both hands now looked slightly swollen and the patient observed that they felt stiff. Both ears were now also of dusky hue and the right external auditory canal (including the drum) presented a faint but definite blush. At this stage, too, an urticarial (½ in.) wheal with a 1 inch hyperaemic periphery appeared on the volar aspect of the left forearm 2 inches proximal to the wrist. Adrenaline, minims 5, was now given subcutaneously and within 3 minutes brought about a noticeable reduction of discoloration of palms and fingers, causing its complete disappearance after some 15 minutes. About 5 minutes after the injection, however, a fresh crop of wheals emerged over the ulnar half of the volar aspect of both forearms, extending from wrist to elbow. These waned at about the same time as the discoloration of the hands. Some 55 minutes after the onset the initial itchy sites were relieved, but itching now commenced in both feet, whence it only disappeared about 10 hours later.

Throughout the entire episode there was a complete absence of nasopharyngeal and oral symptoms, but she showed a slight pallor which disappeared after the injection of adrenaline, as did her earlier somewhat subdued manner, which was changed to talkativeness and a marked tendency to hilarity. Seventy minutes after the introduction of the enzyme-penicillin combination all signs and symptoms, with the exception of the slight itching of the soles of the feet, had disappeared. She was advised to take a further 25 mg. of antihistaminic some 3 hours later as a precaution. Nothing further untoward occurred.

This patient has in the past 'tolerated' 2 courses of parenteral penicillin, as well as many intra-sinal penicillin instillations, with and without hyaluronidase. She is, however, sensitive to one of the sulphonamides, developing 'a swollen upper lip', and also to iodine, the intake of which calls forth a rather fierce iodism.

The purpose of this report is to draw attention to the very unusual occurrence of allergic manifestations during hyaluronidase-penicillin usage intra-sinally. This might conceivably be due to this hydrophilic colloid acting as a 'protective' colloid[1] to the crystalline penicillin in this way slowing down and prolonging its release into the blood stream, thus reducing the chances of the antigen-antibody reaction, and so the likelihood of the allergic manifestations.

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M. Glass.

620 Boston House,
Strand Street,
Cape Town.
3 November 1952.

Psychiatry and General Practice

To the Editor: Dr. Archer is to be congratulated on the stand he has taken as outlined in his letter (this *Journal*, 1 November) wherein *inter alia* he states: 'The proposal to register lay Clinical Psychologists . . . seems a retrograde step'.

Modern concepts of stress and reactions to stress make it abundantly clear that in many psychological disorders there is a background of metabolic, endocrinological or organic mischief such as will not only account for the psychological picture but will lend itself by appropriate therapy to the cure of the symptom-complex.

For instance, myxoedema may present a picture of severe psychological distress which may only be relieved by thyroid therapy. The same therapy has an important niche in the composite adjustment of pan-hypopituitarism in which, while there is thyroid atrophy, there is as a rule no evidence of myxoedema.

Hyperinsulinism presents outward manifestations of disordered psychology and accurate diagnosis is necessary before appropriate therapy can be instituted.

In passing, it cannot be too strongly emphasized that depleted blood sugar is not always due to hyperplasia or tumour of the islets of Langerhans. Portis[1] has made us familiar with the fact that hypoglycaemia is a common finding in anxiety and similar psychological disorders, and that restoration to normality of blood sugar and of nervous balance is possible by vagal blockade with atropine. His finding is illustrative of the additional fact that a condition such as hypoglycaemia arising as the result of stress becomes in itself a stressor factor of considerable degree.

Similarly, a peptic ulcer developing as the result of stress becomes in itself an additional stressor factor and as, moreover, such an ulcer is frequently accompanied by hypoglycaemia,[2] in these circumstances stress becomes of threefold intensity.

Would it be reasonable to expect that a lay psychologist could resolve these problems satisfactorily or that, for instance, his training should enable him to determine accurately that the departure from the normal in the particular case with which he is dealing is the direct result of a subdural haematoma or a cerebral encephalopathy?

Must he be expected to know that in the alcoholic exhibiting at the same time peripheral neuritis and incipient delirium tremens, the apparent organic and functional distortions have a common origin? Unless he is aware that alcohol by reducing cytochrome at the final stage of energy production for the benefit of the cell, thereby deprives the cell of this energy, he cannot know that release of oxygen from administered glucose by B group vitamins will reactivate the energy-producing enzyme and restore the damaged nerve structures to normal.

This serves to remind us all that deprivation of glucose intrinsically or extrinsically and of essential vitamins may determine the emergence of gross neuro-psychiatric disturbance.

The writer has had the opportunity of reading the report produced by Australian and American Medical Officers on individuals who had been submitted to dietary deprivation and physical stress in the Eastern theatre of the last War. One point established beyond all possibility of refutation was that in individuals exhibiting perhaps psychiatric disorder only, there was organic neurological damage present of a progressive and irreversible nature.

The reasons one advances for making reference to these varied conditions mentioned above are threefold.

1. That there is little or no place for lay psychologists, however well trained, in this highly specialized department of medicine.

2. That in allowing themselves to become so highly specialized, our neuro-psychiatrists have drifted away from medical science in general and its ancillary departments of physiology, biochemistry, endocrinology and pathology in particular.

3. That if the accusation embodied in (2) be capable of proof, then the sooner the neuro-psychiatrists, the physicians and surgeons team up in this department, the better for the patient.

In such a team it may be possible to integrate the lay psychologist in a very humble rôle, though one doubts the necessity and the wisdom for such a step. He would be much more usefully employed in laboratory diagnostic research than in the outside field.

For the qualified general practitioner the position is as different as chalk from cheese. He has the knowledge of the essential basic sciences and access to the patient in his home environment. He can, with due instruction and training, become the most important cog in this complicated machine, and specialists in other departments would be well advised to encourage the G.P. to develop his knowledge of the fundamentals of psycho-somatic medicine, as modern research reveals them.

By fostering the integration of the G.P. into this specialized field, benefits of incalculable value must accrue to all concerned, and not the least of these will be made available to the patient.

In recent years there has developed an ever-widening gulf between the ranks of the specialist and the G.P. It is time that steps were taken to close the ranks and, if possible, to interdigitate individual entities. This is a practical way of doing so and one which will redound to the credit of all concerned.

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J. Drummond, M.D., F.R.C.P., Ed.
121 Trust Buildings,
Gardiner Street,
Durban.
10 November 1952.

A REMINDER

To the Editor: Mrs. V., aged 57, with a blood pressure of 210/110 mm. Hg. and with angina of effort, gave a history of irregular vaginal bleeding for about 2 years, and recently of having bled for 5 weeks continuously.

She had a slight cervical erosion which could not be blamed for her discharge. On looking up her previous notes, I found that in 1945 I did a cholecystectomy on her and then felt the presence of small fibroids in the uterus.

My diagnosis therefore was:

1. Fibroids with innocent or malignant degeneration;
2. Carcinoma of the body of the uterus.

Under Pentothal anaesthesia I did a diagnostic curettage, the patient taking the anaesthetic very badly, as expected. To my surprise my curette became entangled with loops of wire which proved to be pieces of a Grafenberg ring. This coiled spring was badly rusted and came out in pieces. An X-ray showed that there were 2 pieces left, but as I could not feel these with a curette or with forceps, I decided on doing a total hysterectomy at a later date.

On interrogating the patient and her husband after the curettage procedure, the following interesting history came to the fore:

Twenty years ago she was told by a doctor that on no account should she have further children, because of her high blood pressure. He forthwith introduced the Grafenberg ring and assured her that she would never fall pregnant again. One year later she gave birth to a boy, now 19 years of age. Mr. and Mrs. V. consequently thought that the ring had either fallen out before pregnancy took place, or would at least have come out when the baby was born. Hence they had put this incident out of their minds and did not mention it to either Dr. Lawson, their house doctor, or to me.

After a week's rest and under protection of Penicillin, I then did the hysterectomy, to find post-operatively, that 2 pieces of this spring had become well embedded in the uterine wall just above the endocervix. This specimen has been sent for pathological investigation, but whether it turns out to be malignant or non-malignant, this case serves as a reminder in 2 directions:

(a) The danger of intra uterine contraceptives;

(b) We must bear in mind the possibility of their having been used in cases of post-menopausal bleeding.

This patient gave us very anxious moments on the operating table because of her high blood pressure and low cardiac reserve, but since then has run a very normal course.

J. D. Joubert, F.R.C.S., F.R.F.P.S., M.B., Ch.B., B.A.
Umtata
10 November 1952.

MELANOPHORE-EXPANDING EFFECT OF ACTH: ITS USE FOR THE BIOLOGICAL ASSAY OF ACTH IN THE SOUTH AFRICAN CLAWED TOAD *XENOPUS LAEVIS*

To the Editor: In view of the obvious need for a simple quantitative test for ACTH in the blood and other body fluids, we have been investigating, during the past few months, its effect in expanding the melanophores of *Xenopus laevis*, the South African clawed toad. We were, therefore, interested to read your views expressed in a recent Editorial (8 November 1952) in which you drew attention to the important and intriguing possibility of the identity of ACTH and intermedin. Since your Editorial may give the impression of a greater unanimity of opinion on this subject than in fact exists, we felt that a more detailed account of the actual present position would be of value to your readers.

The melanophore is a melanin-containing, amoeboid cell present in the skin of amphibia, fishes and reptiles. By altering its shape, and by expanding or contracting black tendrils among the other cells of the animal's skin, it alters the amount of light reflected from the skin surface and hence the apparent colour of the animal.

Hogben (1924) showed that the melanophores of amphibia are under the direct hormonal control of the pituitary gland. Within a wide range of temperature (12—32°C), the melanophores of *Xenopus laevis* expand and the animal becomes dark when placed in the light in a container with a black background, i.e. a surface which absorbs light. The melanophores contract and the frog becomes pale when placed in light on a white background. Hogben and Slome (1931) also showed that, while in Xenopus removal of the anterior lobe abolishes the 'white background' response, removal of both anterior lobe and posterior lobe (which includes the intermediate) abolishes the 'black background' response as well. In the former case the frog remains black on a white background, and in the latter pale on a black background, i.e. the frog shows permanent expansion and contraction of the melanophores respectively. Zondek (1935) showed that the intermediate lobe in amphibia was apparently the primary site of production of this melanophore-expanding hormone, to which the name *intermedin* was given.

Methods of assay of intermedin have been described using a variety of amphibia, the commonest being the frogs *Rana*

temporaria (Europe), *Rana pipiens* (America), and the South African clawed toad *Xenopus laevis*. Landgrebe and Waring (1950) describe a technique using *Xenopus laevis* and consider Xenopus the most satisfactory test animal due mainly to its easy maintenance in the laboratory, even after hypophysectomy. Quantitative results can readily be obtained by a direct estimation microscopically of the degree of expansion of melanophores in the large web of Xenopus. This can be expressed as the melanophore index (Hogben and Slome, 1931). These observations have developed considerable significance for the clinical endocrinologist by the suggestion that intermedin and ACTH are identical. Sprague *et al.* (1950) showed that ACTH manifested appreciable melanophore activity in hypophysectomized frogs. Johnsson and Högberg (1952) reported that injection of adrenocorticotropic hormone into the dorsal lymph sac of *Rana temporaria* produces the same reaction as intermedin, as small an amount as 0.00005 I.U. giving a clearly visible expansion of the melanophores. Sulman (1952a) has independently reported similar chromatophorotropic effects of ACTH on the tree-frog, *Hyla arborea*, and describes a method for the assay of ACTH in blood. These observations have been made in light-adapted frogs which have not been hypophysectomized and it is stated (Editorial, *Lancet*, 1952) that hypophysectomy does not appear essential. It is calculated that as little as 0.001 micrograms of ACTH will cause darkening of the skin in light-adapted frogs.

These reports of the biological effects of ACTH appear to be reinforced by the observations [Johnsson and Högberg (1952), Sulman (1952b)] that chromatophorotropic activity in the blood of human subjects parallels the ACTH output as determined by the adrenal ascorbic-acid-depletion test of Sayers, and shows an increase in Addison's disease, Cushing's syndrome and pregnancy. It is interesting to recall that an increase of skin pigmentation may occur in all these conditions.

An unexplained anomaly in the claims that ACTH and intermedin are in fact the same is that ACTH is a hormone derived from the anterior pituitary gland whereas intermedin is said to come from the pars intermedia. The evidence on extraction of ACTH from intermediate or posterior lobe and of intermedin from anterior lobe is conflicting, but Sulman (1952b) claims that significant amounts of intermedin can be extracted from anterior lobe, and that in primates that have no clearly defined intermediate lobe, intermedin is produced mainly in the anterior lobe. Lock (1952) in a preliminary report, claims that whereas the tree frog, *Hyla arborea*, used by Sulman, responds to both ACTH and intermedin, Xenopus *does not* respond to ACTH and the response to intermedin is not influenced by the presence of ACTH. Since Lock 'separates' ACTH and intermedin by heating the extract to 100°C for 10 minutes with N/10 sodium hydroxide, and since hypophysectomized toads were not used, these experiments cannot be considered as effectively disposing of the value of Xenopus for this assay.

More important objections to the identity of ACTH and intermedin have arisen from the studies on purification of ACTH. Gerschwind, Reinhardt and Li (1952) have shown that separation of ACTH activity (by Sayer's adrenal ascorbic-acid-depletion technique) and of intermedin activity (melanophore-expansion on hypophysectomized *Rana pipiens*) could be achieved by various chemical manipulations, e.g. heating in alkaline solution, use of a discontinuous pH gradient on oxycellulose and by zone electro-phoresis on paper. Morris (1952) also states categorically that ACTH and intermedin are not identical and that the melanophore effect is of no value for the assay of ACTH. He supports his statement on chemical grounds that: (a) Heating with alkali enhances intermedin activity and destroys ACTH activity;

(b) Intermedin can be obtained free from ACTH activity by a carbon absorption process;

(c) ACTH can be obtained free of intermedin activity by counter-current techniques.

Morris concludes that there is little doubt that the melanophore-expanding effects observed in ACTH preparations are due to contamination.

An alternative explanation, which does not seem unlikely and which would reconcile the viewpoints of the biologists and chemists, is that chemical manipulations during purification of ACTH (which incidentally now appears to be a peptide and not a protein), may split off apparent fractions from an ACTH complex with differing physical and chemical properties and biological effects, e.g. adrenal ascorbic-acid-depletion and melanophore-expansion activity.

The finding of increased melanophore-expanding activity in the blood of patients with Addison's disease and Cushing's syndrome and in post-operative and other stress situations is very weighty evidence in support of our alternative explanation. However, some of these experiments have been done on non-hypophysectomized animals. The use of hypophysectomized animals to exclude non-specific stimulation of endogenous intermedin production by substances present in blood and urine extracts appears essential, and since *Xenopus laevis* survives for long periods after hypophysectomy, experiments are being conducted by us to:

(a) Develop an assay method for ACTH concentration in blood and urine using hypophysectomized *Xenopus laevis*, and estimating melanophore-expansion by determination of the melanophore index microscopically in the web;

(b) Study ACTH production in disease states associated with the anterior pituitary and suprarenal glands, and in morbid conditions influenced by ACTH, cortisone and hydrocortisone therapy;

(c) To study the fate of parenterally administered ACTH, and the nature of the systems in blood which destroy ACTH.

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B. M. Bloomberg.
D. Alldis.

Department of Clinical Pathology,
University of Witwatersrand,
Johannesburg.
13 November 1952.

VOTING FOR BRANCH OFFICE-BEARERS

To the Editor: Having to-day received a ballot paper to vote for office-bearers for the Cape Western Branch of the Medical Association, I was impressed with one glaring fact. At least 2 or 3 of the candidates who have allowed their names to go forward for election have not attended a single meeting of the Association for the last 3 or 4 years, as far as I know. As I myself have attended 95% of the meetings, it will surprise me enormously if these gentlemen attended when I was not present.

What I wish to raise is a matter of principle. I think it will be wrong to vote for those members of the Association who never take part in any meetings at all, and I think it is wrong for such doctors to allow their names to go forward. As a matter of principle, members of the Branch Council should show themselves at meetings and it is a pleasure to record that in the past the majority of the members of the Council have attended and can be seen and heard.

Physician.

Cape Town.
14 November 1952.

Transvaalse Provinsiale Administrasie

VAKATURES BY PUBLIEKE HOSPITALE

Aansoeke word ingewag van kandidate met geskikte kwalifikasies vir die onderstaande poste by Publieke Hospitale in die Transvaal.

Aansoeke moet gerig word aan die Geneeskundige Superintendent en Verantwoordelike Geneesheer van die betrokke Hospitaal en moet volle besonderhede bevat aangaande die ouderdom, professionele, akademiese en taalkwalifikasies, ondervinding en huwelikstaat van die applikant en moet voorts 'n aanduiding bevat van die vroegste datum waarop diens aanvaar kan word:

Lewenskostetoelae tans betaalbaar aan voltydse werknemers:

Salaris	*Lewenskostetoelae*	
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Oor £350 ..	£320 p.j.	£100 p.j.

Van persone wat aangestel word, sal verwag word om bevredigende sertifikate in te dien, asook om hulle te onderwerp aan 'n geneeskundige ondersoek by die betrokke hospitaal.

Aansoek vorms is verkrygbaar van enige Transvaalse Publieke Hospitaal of die Provinsiale Sekretaris, Afdeling Hospitaaldienste, Posbus 2060, Pretoria.

Benewens jaarlikse salaris en lewenskostetoelae ontvang voltydse werknemers spoorwegkonsessie en word verlof toegestaan ooreenkomstig die hospitaal verlofregulasies.

Die sluitingsdatum van aansoeke vir die poste is 22 Desember 1952.

Hospitaal	*Vakature*	*Emolumente*	*Opmerkings*
Meerhof, naby Pretoria	Deeltydse Spesialis Geneesheer (1)	£102 10s. per jaar	Half sessie per week. Moet behoorlik deur opleiding en ondervinding gekwalifiseerd wees.
Pretoria	Junior Assistent Geneesheer (1)	£1,200 x 50 —1,500	Geregistreerde mediese praktisyn. Moet behoorlik deur opleiding en ondervinding gekwalifiseerd wees.

(38626)

Practice for Sale

Eastern Cape, dispensing practice in town with recently opened small hospital. Goodwill, drugs, furniture, instruments, at £1,250. Newly installed X-ray equipment for sale separately at £1,150. Roomy surgery rented. Gross income about £2,300. House for sale at £3,000. Owner intends studying further. Write 'A. O. E.', P.O. Box 643, Cape Town.

Wanted

General practitioner required for hospital with 120 beds for 1 February 1953, including medical, surgical, maternity and infectious cases in Native Reserve.

Applications with a complete list of previous experience should be sent to the Secretary, Umlamli Mission Hospital, P.O. Sterkspruit, Herschel District, C.P. Salary according to Cape salary scale.

Partnership Wanted

Married Jewish doctor, aged 30, requires partnership in well-established practice. Commencing 1 February 1953. Write 'A. O. S.', P.O. Box 643, Cape Town.

The Medical Association of South Africa : Die Mediese Vereniging van Suid-Afrika

AGENCY DEPARTMENT : AGENTSKAP-AFDELING

JOHANNESBURG

Medical House, 5 Esselen Street. Telephone 44-9134-5, 44-0817
Mediese Huis, Esselenstraat 5. Telefone 44-9134-5, 44-0817

PRAKTYKE TE KOOP: PRACTICES FOR SALE

(Pr/S34) Progressive Transvaal dispensing practice. Average gross income £3,500 per annum. Excellent surgical facilities. Premium required £2,500 and the following terms could be arranged: £1,250 deposit and the balance over a period of 18 months, starting 3 months after the cash payment. The premium includes drugs, furniture and fittings, estimated at £800. Two transferable appointments worth £230 per annum.

(Pr/S51) Transvaal hospital town dispensing practice. Gross income over £6,000 per annum. It is essential that this practice be worked by two men, one to be a surgeon. Premium required £3,500, and terms could be arranged. Practice can only be sold if house and surgery are bought for cash. Details on application.

(Pr/S54) Established branch practice in Johannesburg. Annual income £1,000. Premium required £500. Very much scope for expansion.

(Pr/S55) Well-established practice in northern suburbs of Johannesburg. Will suit an English-speaking doctor. Premium required £1,000. Full details on application.

(Pr/S60) Prescribing practice in Southern Rhodesia. Monthly income approximately £500. Very modern hospital. Will suit doctor interested in surgery and midwifery. Premium required £5,000, and terms will be accepted.

(Pr/S61) Uitstekende Oos-Transvaalse praktyk, gestig 10 jaar gelede. Die jaarlikse kontantinkomste is oor £3,000. Feitlik geen slegte skuld nie. 'n Deeglike introduksie sal gegee word en is noodsaaklik. Geen premie word gevra nie en die woning is te koop teen £3,000. 'n Verband kan gereël word. Die verkoper wil graag aftree.

PARTNERSHIPS OFFERED

(P/O14) High-class Johannesburg practice. Two partners (one Jewish and one English-speaking) are urgently required for a well-established general practice. Especially men interested in obstetrics. Preference will be given to Johannesburg doctors and men with strong personalities. £2,000 premium required in each case, preferably paid cash. Please apply in writing.

(P/O12) Partnership share in old-established Johannesburg practice. Premium required is £4,000 and liberal terms will be arranged. Please apply in writing.

(P/O10) Old-established firm in large centre in Rhodesia requires two gentile partners as soon as possible. Please apply for full details.

(P/O13) A Jewish partner is required for an excellent Eastern Transvaal dispensing practice. Must be a married man and over thirty years of age, and must have some surgical experience.

* * * *

DURBAN

112 Medical Centre, Field Street. Telephone 24049

PRACTICES FOR SALE: PRAKTYKE TE KOOP

(PD10) General practice, Natal inland city. European and non-European patients. Scope for midwifery and surgery. Premium required £1,250, cash preferred, but terms will be considered. For immediate sale.

(PD13) Natal Lower South Coast practice, near Pondoland border, suitable for retired doctor. Area developing and large Police holiday camp in vicinity. Excellent climate and very good fishing. Premium required £400, includes good stock of drugs and dressings, instruments and dispensary furniture. House for sale £1,800, including stand of one-third morgen. Bond available. For immediate sale. Owner having taken a full-time appointment.

(PD14) Non-European dispensing practice in rapidly expanding industrial and residential area, 11 miles from centre of coastal City. At present no night or after hour calls, no week-end or surgical work undertaken. Practice could be improved if run on a full-time basis, otherwise ideal as a subsidiary practice. Turnover for twelve months ended 31 June 1952 averaged £170 per month. Total expenses including car and travelling expenses, £50 to £60 per month. Premium £750 including drugs, instruments and furniture.

LOCUM REQUIRED

(122) Pondoland. From 1 December 1952 to 30 June 1954. Partnership practice and the senior partner will be remaining in the practice. The partners do not work after 4.30 p.m. during the week and 1 p.m. on Saturday. Mostly Native work. Salary £60–£75 per month, depending on experience, plus free board and lodging, and transport allowance, if locum uses his own car.

(121) Natal South Coast. From 14 December for 5 weeks. Must possess own car. Petrol, oil and servicing allowance will be made. The practice is a mixed one, very little night work. With the exception of two regular trips into the country, the practice is conducted almost entirely within the vicinity. Salary £20 per week.

(116) Near Durban. January 1953. £2 12s. 6d. per day, board, lodging. Own car desirable. Afrikaans essential. Mixed general practice, with R.M.O. appointment.

(119) Northern Natal. 1 January or earlier 26/27 December for one month. £3 3s. 0d. per day, free board and lodging, petrol and oil. Locum must possess own car, £10 car allowance will be made. General mixed practice with mine appointments.

(106) Zululand. From 30 December to 30 January 1953. £2 12s. 6d. per day, car allowance. Single man or woman. Must possess own car. General country practice. Senior partner of the firm will be present throughout living 8 miles away.

(120) Near Durban. From 1 January 1953 for approximately 14 days. £2 12s. 6d. per day, board and lodging and car expenses. Locum should possess his own car. Must be able to dispense as this is a mixed general dispensing practice for non-Europeans only. Not much night work. Suitable for elderly man.

(123) East Griqualand. From 1 January for one month. £2 12s. 6d. per day, free board and lodging and car allowance. Locum must possess his own car. This is a general practice with small R.M.O. and D.S. appointments. Very occasional night and week-end work. No major surgery. One weekly district clinic tour.

* * * *

KAAPSTAD : CAPE TOWN

Posbus 643, Telefoon 2-6177 : P.O. Box 643, Telephone 2-6177

PRAKTYKE TE KOOP : PRACTICES FOR SALE

(1010) Cape Town. Nucleus of practice with excellent scope for expansion. Average annual receipts £1,100. Premium required, £850, which includes drugs, few instruments, half-share furniture. Consulting rooms shared with specialist.

(1016) Eastern Province. Unopposed solus practice. Average annual receipts £2,471. Premium for goodwill £750. Drugs, furniture and instruments offered at £190. Terms available. Attractive modern home to rent at £8 10s. p.m. Rental roomy surgery, £3 p.m.

(992) South-Eastern Cape hospital town. Premium required £1,500, which includes drugs, furniture and instruments worth approximately £1,350. Flat plus surgery to let at £6 p.m. Gross average annual cash takings, £2,500. Easy terms. Owner wishes to specialize.

ASSISTENTE/PLAASVERVANGERS VERLANG ASSISTANTS/LOCUMS REQUIRED

(1167) Namaqualand. 'n Afrikaanssprekende assistent vanaf 1 Januarie 1953. Moterkar word voorsien.

(1168) Boland. So spoedig moontlik tot 22 Januarie. £3 3s. 0d. per dag en vry losies, plus kartoelaag, of kar kan verskaf word. Vrou of man met ondervinding verkieslik.

(1207) Noord-Kaapland. Vanaf 20 Desember vir 1 maand. £3 3s. 0d. per dag, plus losies. Kar word verskaf.

South African Railways and Harbours Sick Fund

APPOINTMENT OF RAILWAY MEDICAL OFFICER: ELSBURG

Applications are invited from registered medical practitioners for appointment to the position of Railway Medical Officer, Elsburg, and for section of railway line Elsburg (inclusive) to Roodekop (inclusive) and to Angus (inclusive), including Palmietfontein Airport, at a salary of £741 per annum, plus £37 10s. per annum transport allowance, plus £30 per annum workshops allowance, plus the fees and allowances prescribed in the Regulations of the Sick Fund, and with the right of private practice.

The salary will be subject to adjustment in accordance with the census of members to be taken on 1 April of each year.

The appointment will be made in terms of the Regulations of the Fund, and will be subject to termination of four months' notice being given by either side.

The successful applicant will be required to reside in the medical district, to take up the appointment on a date to be arranged, and to carry out his duties in accordance with the Regulations of the Fund.

Applications should reach the District Secretary, Western Transvaal District Sick Fund Board, Room 342, Third Floor, New Station Buildings, Johannesburg, not later than 10 January 1953 and should state:

1. Full name.
2. Qualifications (when and where obtained).
3. Experience (when and where obtained).
4. Date of birth.
5. Country of birth.
6. Whether single or married.
7. Whether fully bilingual.
8. Whether South African citizen.
9. What Government appointment, if any, is held.

Canvassing by or on behalf of any applicant is liable to disqualify such applicant.

Any further particulars may be obtained from the District Secretary at the above address, on application.

P. J. Klem
General Secretary

Johannesburg
13 December 1952

Public Service Commission

VACANCIES IN THE PUBLIC SERVICE

1. The attention of medical practitioners, registered with the South African Medical and Dental Council, is drawn to an advertisement appearing in the *Government* and *Provincial Gazettes* of this week, inviting applications for the undermentioned posts:

Post	*Department*	*Salary Scale*
Medical Officer	Health (Johannesburg)	£900 x 50—1,150
District Surgeon, Grade III	Health (Pietermaritzburg, Vereeniging, Bronkhorstspruit and Relief Staff.)	£900 x 50—1,150

2. In addition to salary a cost-of-living allowance at the rate of £320 per annum (married) and £100 per annum, (single) is payable at present.

3. It is emphasized that full and detailed particulars of qualifications and previous experience must be furnished but original certificates and testimonials should not be submitted. Application forms (Z.83 and P.S.C. 8 (*a*)) are obtainable from the Secretary, Public Service Commission, Pretoria, to whom filled-in forms must be addressed.

4. The closing date for the receipt of applications is 27 December 1952. (38532)

Assistant Required

Assistant urgently required as from end January or earlier for big practice on Reef. All surgery done by Principal. Write 'A. O. L.', P.O. Box 643, Cape Town.

Siekefonds van die Suid-Afrikaanse Spoorweë en Hawens

AANSTELLING VAN SPOORWEGDOKTER: NABOOMSPRUIT

Aansoeke word van geregistreerde mediese praktisyns ingewag vir aanstelling in die betrekking van Spoorwegdokter, Naboomspruit, en vir die spoorwegtrajek Middelfontein (insluitend) tot by Drummondlea (uitsluitend) en tot by Zebediela (insluitend), teen 'n salaris van £225 per jaar, plus die gelde en toelaes wat in die regulasies van die Siekefonds voorgeskryf word, en met die reg om privaat te praktiseer.

Die salaris is onderhewig aan wysiging in ooreenstemming met die sensus van lede wat op 1 April van elke jaar afgeneem moet word.

Die dienste sluit in die opmaak van die nodige medisyne wat deur die Fonds voorsien sal word.

Die aanstelling geskied kragtens die regulasies van die Fonds, en opsegging van dienste is onderworpe aan vier maande kennisgewing deur een van beide partye.

Die suksesvolle applikant moet op Naboomspruit woon, op 'n datum wat gereël sal word dienste aanvaar en sy pligte ooreenkomstig die regulasies van die Fonds uitvoer.

Aansoeke moet die Distriksekretaris, Distriksiekefondsraad, Scheidingsstraat, Pretoria, nie later nie as 17 Januarie 1953 bereik, en applikante moet die volgende vermeld:

1. Volle naam.
2. Kwalifikasies (waar en wanneer verkry).
3. Ondervinding (waar en wanneer verkry en opgedoen).
4. Datum van geboorte.
5. Land van geboorte.
6. Getroud of ongetroud.
7. Of ten volle tweetalig.
8. Of Suid-Afrikaanse burger.
9. Watter staatsbetrekking, indien enige, beklee word.

Werwing deur of ten behoewe van enige applikant stel so 'n applikant bloot aan diskwalifikasie.

Enige verdere besonderhede wat verlang word, kan op aanvraag van die Distriksekretaris by bovermelde adres verkry word.

P. J. Klem
Hoofsekretaris

Johannesburg
13 Desember 1952

Motor Industry Sick Benefit Fund

(TRANSVAAL AND ORANGE FREE STATE) PART-TIME MEDICAL OFFICER FOR POTCHEFSTROOM

Applications are invited from fully qualified registered general practitioners in respect of the above-mentioned appointment.

The Fund operates on the closed panel system and the successful candidate will be required to provide consulting room, domiciliary and hospital service (when necessary) for members and their dependants. Further details will be furnished on request.

Applications must reach the Secretary of the Fund, P.O. Box 8477, Johannesburg by Friday 26 December 1952.

18 November 1952

Wanted

Doctor, with 6 years' general practice experience, mainly in Johannesburg, seeks post as an assistant in Johannesburg area with a view to partnership. Write 'A. O. M.', P.O. Box 643, Cape Town.

Assistant Required

Assistant required for partnership practice in Port Elizabeth. Apply with full particulars about previous appointments held and general experience. Duties to commence 1 February 1953. Apply to 'A. O. D.', P.O. Box 643, Cape Town.

O.F.S. Provincial Administration

VOORTREKKER HOSPITAAL KROONSTAD

VACANCY: SPECIALIST ANAESTHETIST

Applications are invited from registered specialist anaesthetists for above post, in a part-time capacity, with right of private practice.

Duties consist of 3 sessions of 4 hours each, per week, at renumeration of £205 per annum per session.

Applications on prescribed form No. Z83, obtainable from the Secretary, or any magistrate's office, together with certified copies of certificates, testimonials, birth certificate, and health certificate, will be received by the undersigned up to 12 noon on Wednesday 31 December 1952.

F. A. van Coller
Medical Superintendent

Kroonstad.
13 November 1952 (A150431)

O.V.S. Provinsiale Administrasie

VOORTREKKER HOSPITAAL KROONSTAD

VAKATURE: SPESIALIS ANAESTETIKUS

Aansoeke word gevra van geregistreerde spesialis anaestetikusse vir bogenoemde pos, in 'n deeltydse hoedanigheid met die reg van private praktyk. Dienste bestaan uit 3 sessies van 4 uur elk, per week, teen besoldiging van £205 per jaar per sessie.

Aansoeke op die voorgeskrewe vorm Z83, verkrygbaar van die Sekretaris of enige magistraatskantoor, tesame met gesondheid- en geboortesertifikate, sowel as gesertifiseerde afskrifte van sertifikate en getuigskrifte, sal deur ondergetekende ontvang word tot 12 nm. op Woensdag 31 Desember 1952.

F. A. van Coller
Geneesheer-Direkteur

Kroonstad
13 November 1952 (A150431)

South African Railways and Harbours Sick Fund

APPOINTMENT OF RAILWAY MEDICAL OFFICER: KLERKSDORP 'A'

Applications are invited from registered medical practitioners for appointment to the position of Railway Medical Officer, Klerksdorp 'A', at a salary of £716 per annum, plus a surgical allowance of £100 per annum, plus the fees and allowances prescribed by the Regulations of the Sick Fund, and with the right of private practice.

The salary will be subject to adjustment in accordance with the census of members to be taken on 1 April of each year.

The appointment will be made in terms of the Regulations of the Sick Fund, and will be subject to termination on four months' notice being given by either side.

The successful applicant will be required to reside at Klerksdorp, to take up the appointment on a date to be arranged, and to carry out his duties in accordance with the Regulations of the Sick Fund.

Applications should reach the District Secretary, Cape Northern District Sick Fund Board, Florence Road, Kimberley, not later than 2 January 1953, and should state:

1. Full name.
2. Qualifications (when and where obtained).
3. Experience (when and where obtained).
4. Date of birth.
5. Country of birth.
6. Whether married or single.
7. Whether fully bilingual.
8. Whether South African citizen.
9. What Government appointment, if any, is held.

Canvassing by or on behalf of any applicant is liable to disqualify such applicant.

Any further particulars may be obtained from the District Secretary at the above address, on application.

P. J. Klem
General Secretary

Johannesburg
13 December 1952

Siekefonds van die Suid-Afrikaanse Spoorweë en Hawens

AANSTELLING VAN SPOORWEGDOKTER: KLERKSDORP „B'

Aansoeke word van geregistreerde mediese praktisyns ingewag vir aanstelling in die betrekking van Spoorwegdokter, Klerksdorp „B', and vir die spoorwegtraject Harrisburg (insl.), tot by Koekemoer (insl.) en tot by Hartebeestfontein (insl.) teen 'n salaris van £781 per jaar, plus die gelde en toelaes wat in die regulasies van die Siekefonds voorgeskryf word, en met die reg om privaat te praktiseer.

Die salaris is onderhewig aan wysiging in ooreenstemming met die sensus van lede wat op 1 April van elke jaar afgeneem moet word.

Die aanstelling geskied kragtens die regulasies van die Siekefonds, en opsegging van dienste is onderworpe aan 4 maande kennisgewing deur een van beide partye.

Die suksesvolle applikant moet in Klerksdorp woon, sy eie spreekkamers voorsien in die Westelike gebied van Klerksdorp, diens aanvaar op 'n datum wat gereël sal word, en sy pligte ooreenkomstig die regulasies van die Siekefonds uitvoer.

Aansoeke moet die Distriksekretaris, Distriksiekefondsraad, Florenceweg, Kimberley, nie later nie as 2 Januarie 1953 bereik, en applikante moet die volgende vermeld:

1. Volle naam.
2. Kwalifikasies (waar en wanneer verkry).
3. Ondervinding (waar en wanneer verkry en opgedoen).
4. Datum van geboorte.
5. Land van geboorte.
6. Getroud of ongetroud.
7. Of ten volle tweetalig.
8. Of Suid-Afrikaanse burger.
9. Watter staatsbetrekking, indien enige, beklee word.

Werwing deur of ten behoewe van enige applikant stel so 'n applikant bloot aan diskwalifikasie.

Enige verder besonderhede wat verlang word, kan op aanvraag van die Distriksekretaris by bovermelde adres verkry word.

P. J. Klem
Hoofsekretaris

Johannesburg
13 Desember 1952

For Sale

An Anaesthetic Machine, Boyles, G. Type. It has been in use under a year and is in excellent condition. Microscope, with dark background, Bausch and Lomb, monocular vision, with oil immersion. Several medical books, a list of which could be obtained on inquiry. Write 'A. O. P.', P.O. Box 643, Cape Town.

For Sale

Prescribing practice, centre of Cape Town, income about £1,500 a year; several appointments can be transferred. Sale includes X-ray machine, diathermy, furniture, etc. For further details write 'A. O. U.', P.O. Box 643, Cape Town.

Printed by Cape Times Ltd., Parow, and Published by the Proprietors, THE MEDICAL ASSOCIATION OF SOUTH AFRICA, MEDICAL HOUSE, 35 Wale Street, Cape Town. P.O. Box 643. Telephone 2-6177. Telegrams: 'Medical'